

**Into the Mouths of Babes: Hyperactivity, Food Additives and the
History of the Feingold Diet**
[volume 1 of 2 volumes]

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Abstract

In 1974 Random House published a popular and controversial book entitled *Why Your Child is Hyperactive*. The author, San Francisco allergist Ben F. Feingold, claimed that hyperactivity was caused by food additives and was best prevented and treated with a diet, subsequently dubbed the 'Feingold diet', free of such substances. Reaction to the idea was swift. The media and parents found Feingold's environmentally-based theory intriguing, as it provided an aetiological explanation for hyperactivity that was both sensible and topical. The medical community, in contrast, was suspicious and designed double-blind trials to test his theory. The dominant perception emerging out of these tests was that Feingold's hypothesis was incorrect and, soon after Feingold's death in 1982, medical and media attention faded away.

Drawing on unpublished archival material, medical literature, popular media sources and oral history interviews, this thesis explores the rise and fall of the Feingold diet. It examines the origins of Feingold's idea, the manner in which his theory was disseminated to the medical community and the broader public, and analyses how physicians and patients evaluated whether or not Feingold's hypothesis was correct. Aiming to contribute to the histories of allergy, psychiatry and nutrition, the thesis contends that social factors, rather than scientific testing, were largely responsible for the fate of the Feingold diet. Some of these factors include Feingold's methods and approach to describing and promoting his diet, the professional and economic interests of medical practitioners and the food, chemical and pharmaceutical industries, and the difficulties inherent in following the diet. From a broader historiographical perspective, the history of the Feingold diet suggests that in order to understand how medical controversies are resolved it is essential to analyse the historical context within which they emerge.

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Abbreviations

AAA	American Academy of Allergy
AAAAI	American Academy of Allergy, Asthma and Immunology
AAP	American Academy of Pediatrics
AASA	American Association for the Study of Allergy
ACA	American College of Allergists
ACSH	American Council on Science and Health
ADD	Attention-Deficit Disorder
ADHD	Attention-Deficit/Hyperactivity Disorder
ADI	Allowable Daily Intake
<i>AJP</i>	<i>American Journal of Psychiatry</i>
AMA	American Medical Association
APA	American Psychiatric Association
BBC	British Broadcasting Corporation
BHA	Butylated Hydroxyanisole
BHT	Butylated Hydroxytoluene
<i>BMJ</i>	<i>British Medical Journal</i>
BSE	British Bovine Spongiform Encephalopathy
CBC	Canadian Broadcasting Corporation
CHADD	Children and Adults with Attention Deficit/Hyperactivity Disorder
CJD	Creutzfeldt-Jakob Disease
CMA	California Medical Association
CNN	Cable News Network
CSPI	Center for Science in the Public Interest
DES	Diethylstilbestrol

<i>DSM</i>	<i>Diagnostic and Statistical Manual of Disorders</i>
EEG	Electroencephalogram
FAUS	Feingold Association of the United States
FDA	Food and Drug Administration (United States)
FSA	Foods Standards Agency (United Kingdom)
IgE	Immunoglobulin E
<i>IJSP</i>	<i>International Journal of Social Psychiatry</i>
IUD	Intrauterine Device
<i>JAACP</i>	<i>Journal of the American Academy of Child Psychiatry</i>
<i>JAMA</i>	<i>Journal of the American Medical Association</i>
K-P	Kaiser-Permanente
LSD	Lysergic Acid Diethylamide
MBD	Minimal Brain Damage
MBD	Minimal Brain Dysfunction
MCS	Multiple Chemical Sensitivity
MMR	Measles, Mumps and Rubella Vaccine
MSG	Monosodium Glutamate
NACHFA	National Advisory Committee on Hyperkinesis and Food Additives
NDEA	National Defense Education Act
NIH	National Institutes of Health
PTA	Parent-Teacher Association
PTSD	Post Traumatic Stress Disorder
SSAAC	Society for the Study of Asthma and Allied Conditions
USDA	United States Department of Agriculture

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Chapter 1

Introduction

In 1974 a self-help book written by Ben F. Feingold (1899-1982)¹ entitled *Why Your Child is Hyperactive* arrived on the shelves of book stores across North America.² On the surface, the Random House publication was not particularly exceptional. By the mid-1970s hyperactivity, a disorder characterised by hyperactive, impulsive, inattentive, aggressive and defiant behaviour, was the most commonly diagnosed childhood psychiatric disorder.³ Many other books, including primers, self-help books and medical textbooks had also been written about the disorder. Medical journals, such as the *American Journal of Psychiatry (AJP)*, the *Journal of the American Academy of Child Psychiatry (JAACP)* and *Pediatrics*, had published hundreds of articles on the disorder and the pharmaceutical companies, such as Ciba, that advertised on their pages made significant profits on the sales of hyperactivity drugs such as methylphenidate, better known by its trade name, Ritalin. The popular American magazine *Life* had also published a seven-page article on hyperactivity in October of 1972. Perhaps most indicative of the emergence of hyperactivity as a disorder of both medical and social significance was the publication of two books, Peter Schrag and Diane Divoky's *The Myth of the Hyperactive Child: And Other Means of Child Control* (1975) and Peter Conrad's *Identifying Hyperactive Children: The Medicalization of Deviant Behavior* (1976), which critiqued the validity and meaning of the disorder. While the preface to Schrag

¹ In most publications Feingold's birth is listed as 1900; indeed, this is the year in which Feingold thought he was born. The recent discovery of a census document, however, indicates that he was actually born on 9 June 1899. The link to this document is found here: www.feingold.org/pg-aboutus.html.

²

³ Hyperactivity is used throughout to denote what physicians now call Attention-Deficit/Hyperactivity Disorder (ADHD), partly because it was the most common term for the disorder during the 1970s, but also because it continues to be the term most patients, parents and physicians recognize and understand. Hyperactivity has been otherwise known as minimal brain damage, minimal brain dysfunction, acting out, hyperkinesis and Attention Deficit Disorder (ADD). The disorder also shared similarities to other childhood disorders, including Learning Disorder and Oppositional Conduct Disorder. For a study on the prevalence of hyperactivity during the 1970s, see:

and Divoky's book stressed that hyperactivity was 'not a genuine disorder or disturbance, but a label which provides comfort to parents, teachers and maybe other school pupils' and a 'moral panic ... whipped up by increased school indiscipline and the pharmaceutical industry', Conrad was concerned that the medicalisation of such behaviour meant that the social origins of hyperactivity were overlooked.⁴

Feingold's *Why Your Child is Hyperactive* was similarly contentious, but in a strikingly different way. Unlike psychoanalysts who claimed that hyperactivity was due to unresolved family conflict, social psychiatrists who thought that socio-economic problems were to blame or the increasing number of biological psychiatrists who suspected that hyperactivity was caused by neurological dysfunction, Feingold, a well-known San Francisco allergist, argued that the ingestion of food additives caused such behaviour and that the disorder could be alleviated with the food additive-free Feingold diet.⁵ Almost immediately Feingold's hyperactivity hypothesis fomented controversy. Spurred by media reports, Feingold's books and word of mouth, thousands of parents tried the diet and discovered that it appeared to ease the symptoms experienced by their children. Some were so convinced of the Feingold diet's efficacy that they founded Feingold Associations across North America and elsewhere which promoted the diet, developed lists of 'Feingold-friendly' foods and provided support to member families.⁶ The media also picked up on Feingold's story during the 1970s and featured him and his diet on popular American television programmes such as *Today* and the *Phil Donahue Show*, in influential newspapers such as the *New York Times*, and in widely circulated magazines such as *Newsweek*.⁷ Shortly before the publication of *Why Your Child*

⁴ Peter Conrad, *Identifying Hyperactive Children: The Medicalization of Deviant Behavior* (Toronto: Lexington Books, 1976), 1-6.

⁵ Weinreb and R. M. Counts, 'Impulsivity in Adolescents and Its Therapeutic Management', *Archives of General Psychiatry* 2 (1960), 548-58, at pp. 549-50; E. A. Grootenboer, 'The Relation of Housing to Behavior Disorder', *American Journal of Psychiatry* 119 (1962/1963), 469-72, at p. 471;

⁶
⁷

is *Hyperactive*, Senator Glen Beall of Maryland included Feingold's findings about the perils of food additives in the U.S. Congressional Record. Due in part to this attention, *Why Your Child is Hyperactive*, as well as Feingold's sequel, *The Feingold Cookbook for Hyperactive Children*, which was co-written by Feingold's wife, Helene S. Feingold, became best sellers, the latter reaching fourth place on the *New York Times* non-fiction bestseller's list.⁸ Despite such intense media and political spotlight, popular interest in the Feingold diet had faded by the mid-1980s, especially after its founder's death in 1982. Nevertheless, the Feingold Association of the United States (FAUS) continued to promote the diet and attract members.

In contrast to the public's enthusiasm, most physicians, including psychiatrists, paediatricians and allergists, were sceptical about Feingold's hypothesis and designed numerous trials to test it, most occurring between the publication of *Why Your Child is Hyperactive* in 1974 and Feingold's death in 1982. The majority of physicians claimed that the findings of these trials disproved Feingold's hypothesis, and were reluctant to recommend his diet to treat hyperactive children. As a result, both Feingold's hypothesis and his diet were marginalised to the fringes of medical practice. Today, most physicians concur that the Feingold diet was never proven to be effective and consider it a regrettable, yet persistent, aberration in the progression of treatment for hyperactivity.⁹

Close examination of the trials intended to test Feingold's hypothesis, as well as the context in which they were designed and conducted, however, reveals that, as a whole, they yielded neither uniformly positive nor negative findings.¹⁰ While some trials were supportive of

⁸ Proceeds from *The Feingold Cookbook for Hyperactive Children* were used as an endowment for FAUS. Letter from Ben. F. Feingold to Beatrice Trum Hunter, 2 October 1979, from the Beatrice Trum Hunter Collection, Howard Gotlieb Archival Research Center at Boston University, Box 47;

⁹ Philippe A. Eigenmann and Charles A. Haeggeli, 'Food Colourings and Preservatives: Allergy and Hyperactivity', *Lancet* 364 (2004), 823-4; C. R. Steer, 'Managing Attention Deficit/Hyperactivity Disorder: Unmet Needs and Future Directions', *Archives of Disease in Childhood* 90 (2005), i19-i25, at p. i22;

¹⁰ A recent review which examined 15 trials of the Feingold diet concluded that, when analysed statistically, the trials as a whole indicated that food additives did increase the hyperactivity of children already diagnosed with the disorder. The reviewers encouraged their colleagues to conduct more research into Feingold's thesis.

Feingold's conclusions, others were negative and still others reported mixed results. Most articles concluded with the suggestion that more research into Feingold's claims was necessary and, accordingly, the small numbers of trials conducted in the last twenty years have been more supportive of Feingold's thesis.¹¹ Why, then, were most physicians so quick to reject the Feingold diet? Conversely, why were thousands of families willing to discount official medical opinion regarding the Feingold hypothesis and try the diet for themselves? What explains the rise and fall of the Feingold diet?

In order to answer these questions, this thesis investigates the history of the Feingold diet, including its origins and place within the contemporary contexts of allergy and psychiatry, its promotion by Feingold and his supporters and its reception by physicians, the families of hyperactive children and the media. The aim of the thesis is to provide a contextual account of the history of the Feingold diet which not only contributes generally to the histories of allergy and psychiatry, and specifically to the history of hyperactivity, but also engages with challenging and topical issues within the history of medicine. Such issues include the development, dissemination and evaluation of novel medical ideas, the role of unconventional medical theories and practices, as well as the place of controversial disciplines, such as allergy and psychiatry, within medicine and the manner in which patients and their families interpret alternative and orthodox medical advice and make decisions about their treatment.

While such reviews are provocative and might prompt some physicians to reconsider the Feingold diet, they mistakenly suggest that all physicians make decisions about novel theories by analysing the available trials in a similarly critical and statistically sound manner. It is not likely that clinicians or medical researchers during the 1970s and 1980s, before the era of the internet, had access to more than a few of the trials reviewed; the trials were published not only in American journals, but also Australian and Canadian journals, a monograph and a PhD dissertation. Moreover, the results of most of the trials could be interpreted in different ways. Such reviews indicate that it is difficult to draw broad conclusions about the Feingold diet based on the trials designed to test.

The argument set out in this thesis is that specific socio-economic and political factors, rather than the rigours of scientific testing, were paramount in determining whether or not physicians and patients would implement the Feingold diet. Such factors included Feingold's methods, personality and, particularly, the manner in which he depicted the genesis of his theory and subsequently promoted it. Although the medical community subjected Feingold's theory to scientific trials, such tests were beset by problems related to design, methodology and interpretation. Both critics and supporters of the Feingold diet ignored or exploited these flaws in order to support their own positions. Such actions suggest that economic, political and ideological factors, such as ties to pharmaceutical and chemical companies, the threat Feingold's hypothesis posed to prevailing hyperactivity theories and treatments and prevailing beliefs about the links between nutrition, allergy and behaviour, influenced physicians more than the results of the trials. Finally, the families who contemplated employing the Feingold diet to treat their children's hyperactivity were swayed by social, domestic and economic pressures, as well as their particular beliefs about the danger of chemicals in the environment and the wisdom of prescribing psychoactive drugs to children, rather than the opinions of their physicians. For those who attempted the diet, practical barriers, including problems accessing and preparing food additive-free meals, preventing violations of the diet and accurately recording reactions to various foods, meant that, even if parents found the Feingold diet to be effective in theory, it was often onerous to implement in practice.

Given these arguments, the thesis is framed around three themes, all of which reflect key areas of debate within the field of medical history. Each theme represents a major section of the thesis which has been divided into smaller chapters. The first theme developed involves the origins of the Feingold diet and addresses how physicians described the evolution of their medical theories, as well as the impact these narratives had on whether physicians and

patients accepted or rejected their ideas. In order to provide a historical context for the development of Feingold's hypothesis, Chapter 2 analyses the history of hyperactivity, the roots of which are a matter for debate. Although physicians sought to portray hyperactivity as dating back to the nineteenth-century, thus delineating the disorder as a universal, timeless condition, Chapter 2 contends that such interpretations are misguided, and that the roots of hyperactivity are centred instead in the Cold War politics of the late 1950s.

Feingold's depiction of the origins of his theory, which is analysed in Chapter 3, were similarly constructed in a manner that would lend credit to his idea. Feingold's theory about hyperactivity spanned the seemingly disparate medical disciplines of psychiatry and allergy, two disciplines which struggled for legitimacy, continually courted controversy and yet purported to investigate questions crucial to the medical understanding of humanity: namely, how to explain the nature of human emotional experience and how the body distinguishes between the harmful and innocuous substances to which it is exposed in the environment.¹² Because these medical questions also had philosophical, ideological and political ramifications, Feingold presented the origins of his theory in a careful, discreet manner which aligned it with the ideas of certain physicians and scientists and not others. In doing so, he described a history of his diet which was detached from the broader histories of food allergy and hyperactivity and, more specifically, from previous notions linking food allergy and mental health problems such as hyperactivity. Chapter 4 examines the history of food allergy and analyses why Feingold, in an attempt to convince orthodox allergists that his hypothesis was valid, chose to disassociate his theory from the controversial views of food allergists.

The second section examines how Feingold's theory was disseminated to physicians and the public and addresses how the manner in which novel medical notions were communicated to

¹² Psychiatry's struggles for legitimacy, especially in North America, have been described by many historians.

the medical and lay communities affected the reception of these ideas. It also explores how the response to new medical theories depended not only on how valid or well-supported such ideas were, but also how well they intersected with contemporary concerns and ideologies.¹³ Chapter 5 examines how, although Feingold originally attempted to promote his idea to the medical community, initially presenting it to the American Medical Association (AMA) conference in 1973, he had difficulty publishing his theory in leading medical journals. Eventually, he shifted his efforts to the popular press and to communicating his ideas directly to parents either in person or via the media. Although this change in strategy ensured that his ideas became well-known, it had ramifications for how the medical community perceived both him and his hypothesis. Concurrently, public interest in the Feingold diet was fuelled by both contemporary concerns about the effects of food additives on health and dissatisfaction with conventional explanations and treatments for hyperactivity. While Chapter 6 explores the debates about how to understand and deal with hyperactivity, and demonstrates how such controversy helped convince parents to seek alternatives such as the Feingold diet, Chapter 7 explores contemporary concerns about food additives and how these also fuelled interest in theories linking chemicals in food to behavioural disturbance.

¹³ Among the many historical explorations of this topic, the following examples are particularly pertinent in that they discuss ideas that were disseminated to both physicians and the general public. The ‘seed and soil’ metaphor explored by Michael Worboys in relation to germ theory could also be extended to the reception of other medical ideas. Just as germs were thought to require the correct ‘soil’, found in human bodies, in order to thrive, the ‘seeds’ of medical theories demand the correct social, ideological and cultural environment in order to germinate. Edward Shorter, *From Paralysis to Fatigue: A History of Psychosomatic Illness in the Modern Era* (New York: The Free Press, 1992); Emily Martin, *Flexible Bodies: Tracking Immunity in American Culture from the Days of Polio to the Age of AIDS* (Boston: Beacon Press, 1994); David Healy, *The Antidepressant Era* (Cambridge, MA: Harvard University Press, 1997); Nancy Tomes, *The Gospel of Germs: Men, Women and the Microbe in American Life* (Cambridge, MA: Harvard University Press, 1998); Michael Worboys, *Spreading Germs: Disease Theory and Medical Practice in Britain, 1865-1900* (New York: Cambridge University Press, 2000); Sheila M. Rothman and David J. Rothman, *The Pursuit of Perfection: The Promise and Perils of Medical Enhancement* (New York: Pantheon Books, 2003); Rima D. Apple, ‘The More Things Change: A Historical Perspective on the Debate Over Vitamin Advertising in the United States’, in John W. Ward and Christian Warren (eds.) *Silent Victories* (Oxford: Oxford University Press, 2007), 193-206; Roberta Bivins, *Alternative Medicine? A History* (New York: Oxford University Press, 2007); David Herzberg, *Happy Pills in America: From Miltown to Prozac* (Baltimore: Johns Hopkins University Press, 2009).

The third strand of the thesis explores how physicians, the media and parents interpreted conflicting reports about Feingold's hypothesis and came to conclusions about it. Chapter 8 turns to how Feingold's theory was presented in the media and suggests that, rather than attempting to influence the debate about the Feingold diet, the media's response reflected not only how complex debates about Feingold's hypothesis were, but also the deep divisions in American society with regards to both food additives and hyperactivity, as well as the government's role in regulating the food and drug industries. As such, the manner in which the Feingold diet was discussed in the popular press provides insight into how controversial medical theories were debated and discussed during the 1970s and early 1980s in American media.

The process by which physicians made decisions about Feingold's theory is examined in Chapter 9. Analysis of the double-blind controlled tests of the Feingold diet reveals substantial problems in how they were designed and interpreted. Nevertheless, both Feingold's supporters and detractors claimed that these trials provided strong evidence to support their particular positions. In order to explain these discrepancies, it is argued that the most important challenge faced by new and, especially, iconoclastic medical theories was not necessarily those posed by scientific trials, but instead the process of integrating into, or perhaps questioning, the prevailing ideologies, power structures and economic systems that have upheld existing authoritative medical knowledge. Despite mixed results based on poorly-designed trials, most physicians believed that the Feingold diet had been proven to be invalid, suggesting that decisions about novel medical theories are based on more than scientific testing alone.

Although parents have relied on physicians for advice about their children's health, it has largely been up to them to make decisions about the medical treatment their children receive.

Indeed, more than medical approval is required for proposed psychiatric treatments to become mainstream medical practice. Patients and their families have had to trust that the tangible benefits of treatment are worth their costs. It took considerable time for parents employing the Feingold diet to identify food additive-free products in the supermarket, monitor what their hyperactive children consumed, and convince them not to violate the diet by sneaking candy, ice cream, or coloured drinks. Successful execution of the Feingold diet required patience, perseverance, and willingness to defy mainstream medical opinion, and was complicated by the fact that other remedies, most notably stimulant medication, was available, inexpensive, simple and appeared to be effective. Parents also had to rely on their children to comply with the diet in order for it to work. Although children were able to circumvent the restrictions of the diet at friends' houses, at school and at the corner store, they also faced considerable internal and external pressure to achieve academically, control their behaviour and forge positive relationships with classmates, teachers and relatives. In order to unravel such complex circumstances, the oral histories of adults who experienced the Feingold diet as children have been included, as well as those of their parents. Chapter 10, which relies chiefly on oral history evidence, therefore, describes why families chose to employ the Feingold diet, how they dealt with the stresses of planning food additive-free meals and whether or not they found the diet to be effective.

The necessity of convincing children that the Feingold diet would be helpful was yet another example of the difficulties faced by Feingold families. Indeed both detractors and supporters of the diet described the regime as challenging, time-consuming and expensive.

Nevertheless, the Feingold diet is still promoted by FAUS and attracts supporters. Moreover, in the wake of increased concern about nutrition and health in the late 1990s and early 2000s, for example, the Bovine Spongiform Encephalopathy (BSE) crisis in the Britain and elsewhere, the organic food movement and concern about obesity in children, interest in the

diet and the link between nutrition and mental health developed once again. Chapter 11, the conclusion of the thesis, examines the Feingold diet's endurance and examines why interest in Feingold's idea has increasingly shifted from the United States to Britain.

Sources

In order to develop these themes comprehensively, a wide range of primary sources have been consulted, reflecting the views of physicians, patients and the media regarding the Feingold diet. The first category of sources focuses on Feingold's academic and popular publications, correspondence and speeches. Feingold's writings about hyperactivity spanned a twenty-five year period, the second half of his medical career, in which he published widely about bronchial allergy disease, flea bite allergies, psychosomatic allergy and clinical allergy practice. From these publications it is possible to outline the theoretical framework which structured much of Feingold's thinking about diet and hyperactivity, and helps to explain the manner in which he depicted the origins of his diet. Supplementing Feingold's writing are the publications of FAUS, which took up Feingold's cause after his death. FAUS not only provided lists of Feingold-friendly food to parents, but it also recorded patients' success stories, cited common challenges to persevering with the diet, warned parents about other chemical dangers to their children's health, provided the first point of contact for parents seeking alternative treatments for hyperactivity and defended the Feingold diet from its numerous critics.

Finally, archival evidence from the Archives of the American Academy of Allergy, Asthma and Immunology (AAAAI), the Beatrice Trum Hunter archive at Boston University and the Theron Randolph archive at Harvard University have also been used to discover how allergists, clinical ecologists and lay experts discussed Feingold's theory and ideas about food allergy in general. Included in these archives are correspondences between physicians,

patients, lay experts and medical associations, such as the AAAAI and the AMA, as well as the meeting minutes of committees relating to topics such as food allergy and clinical ecology. The unpublished archival evidence not only indicates how individuals felt about the Feingold diet, but also reveals how his idea quickly became a contentious issue for both allergists and the public.

The second category of primary sources utilised serves to represent the opinions of the medical community, especially allergists, psychiatrists and paediatricians, regarding the Feingold diet. The medical journals consulted include, but are not limited to, the *Annals of Allergy*, the *Journal of Allergy*, *AJP*, *Lancet*, *Pediatrics*, the *Journal of the American Medical Association (JAMA)* and *Archives of Disease in Childhood*, and span the early twentieth century to the present day. In particular, accounts of clinical case studies, double-blind trials and book and literature reviews have been analysed, as well as editorials, letters to the editor and official position statements, in order to trace the debates surrounding food allergy generally and the Feingold diet in particular. Clinical allergy textbooks, such as P. G. H. Gell and R. R. A. Coombs' *Clinical Aspects of Immunology*, Albert Rowe's *Food Allergy* and Jonathan Brostoff and Stephen Challacombe's *Food Allergy and Intolerance*, as well as allergy primers and handbooks for people without medical training, also constitute important primary source material.¹⁴

Media reports about the Feingold diet, food additives, hyperactivity and other related subjects provide another key source of evidence. While many of the media sources represent some of the major American newspapers and magazines, including the *New York Times*, *Washington Post* and *LA Times*, smaller newspapers and magazines have also been consulted to see how issues related to the Feingold diet were reported in the various regions of the United States.

North American and British internet media sources, such as the websites of the Cable News Network (CNN), the British Broadcasting Corporation (BBC) and the Canadian Broadcasting Corporation (CBC) have also been used to explore more recent developments involving Feingold's hypothesis. Media sources have been analysed critically to reflect not only how the media represented the Feingold diet to the public but also to examine the manner in which journalists, medical columnists, editors and letter writers interpreted the controversy that surrounded Feingold's idea and linked it to other contemporary concerns and debates.

Oral history records of both patients and physicians, and supporters and critics, of the Feingold diet represent the final category of primary source material. The primary reason for the inclusion of oral history in this thesis is simply that the voices of patients are rarely documented in typical archival records and, therefore, many medical historians are unable to reflect adequately patients' emotions, opinions and reflections regarding their experiences, not to mention the process by which they interpreted the diagnoses and treatments provided to them by their physicians. As oral historians Paul Thomson and Rob Perks have remarked, oral history can 'create a more complex and rounded picture of the past', 'uncover the hidden informal culture of work', and explore 'health as experienced by the ... patient rather than the ... doctor'.¹⁵

Interviews of forty-one patients, parents, medical researchers and clinicians have been included in this project. While the oral history interviews often represent the only evidence available that captures the perspectives of patients and parents, in the case of physicians, such evidence supplements what they have published and often serves to enhance and refine these sources. The interviews conducted were semi-structured, but also open-ended, in that a standardised list of questions was used to frame the interview, but the interview was not

limited to these questions. Although most interviews were conducted over the telephone, others were conducted via email or in person. Once the transcript of the interview was analysed, interviewees were often sent follow-up questions to clarify or elaborate responses. Oral history provides the primary evidence for Chapter 10, but also supplements the documentary evidence employed in other chapters. A number of interviewees chose to remain anonymous and measures have been put in place to ensure their anonymity. Instead of assigning pseudonyms to these participants, anonymous interviewees are only distinguishable from one another in footnote citations and in the bibliography by the type of interview in which they participated and the date of their interview. In cases where two family members were interviewed and only one of them wanted to maintain anonymity, both interviewees have been made anonymous so as not to reveal either person's identity.¹⁶

Complementary Histories: Allergy and Psychiatry

The wide range of primary sources employed to investigate the history of the Feingold diet is mirrored in the variety of secondary sources which have informed the methodology of this thesis. The history of the Feingold diet bridges what could be seen as a rather large gulf between the histories of allergy and immunology, and the history of psychiatry. While Feingold's background as an allergist influenced his theory of hyperactivity, the disorder itself was typically viewed as a psychiatric problem and it had been psychiatrists who had dominated investigations into its aetiology and treatment. As such, historical research on both allergy and psychiatry, as well as work exploring the histories of nutrition, childhood and the interface between medical science and the public, have been relied upon not only to inform the methodologies employed in the thesis, but also to provide contexts for Feingold and his theory. How have historians characterised the worlds of allergy and psychiatry that

¹⁶ All interviewees were given the option of anonymity and have signed consent and copyright forms in accordance with the University of Exeter's Ethics Committee.

Feingold attempted to straddle? What factors have shaped their respective development and philosophical underpinnings and how might these have affected the reception of the Feingold diet? How have allergy and psychiatry dealt with radical theories such as Feingold's? In examining how historians of allergy and psychiatry have answered these questions it is possible to place Feingold and his theory into a relevant medical context and better understand the professional milieu in which his diet had to survive.

Examining the histories of allergy and psychiatry superficially, it is difficult to think of two more different historiographies, both in terms of quantity of material and general style of writing. The history of allergy is a new field in which Mark Jackson's *Allergy: The History of a Modern Malady* and Gregg Mitman's *Breathing Space: How Allergies Shape our Lives and Landscapes* are the only monographs written on the subject.¹⁷ Likewise, there are only a small number of books and articles written about related topics such as asthma and clinical ecology, as well as the broader category of immunology. This is despite the fact that twenty Nobel prizes have been awarded for studies related to allergy and immunology.¹⁸

Although historians and immunologists have reportedly engaged in heated debate at conferences concerning aspects of the history of immunology, by the mid-1990s, Warwick Anderson, Miles Jackson and Barbara Gutmann Rosenkrantz had detected 'a sense of intellectual inevitability' or progressivism that was filtering through the historiography of immunology, suggesting that the historians had 'worked largely within the conventional boundaries, or "invented traditions", established by immunologists themselves'.¹⁹ Although

¹⁷ A handful of historical articles have been written about allergy, many of them in a 2003 volume of *Studies in History and Philosophy of Biological and Biomedical Sciences*.

¹⁸ In contrast, only two Nobel Prizes can be said to have been awarded for psychiatric research. In 1927 Julius Wagner-Jauregg (1857-1940) was awarded the prize in medicine for his research into using fever to cure psychoses. Egas Moniz (1874-1955) shared the award in 1949 for his work on lobotomy (leucotomy). Although most contemporary psychiatrists might feel inclined to disassociate themselves from this latter award, Jack Pressman's book on the history of lobotomy helps explain why the procedure became so popular.

¹⁹ . Alberto Cambrosio, Peter Keating and Alfred Tauber reported that at a meeting in Ischia in 1993, scientists and historians clashed over who was to interpret the history of immunology and how it 'seemed to

the studies by Jackson, Mitman, Carla Keirns and others during the last few years have served to change this tendency, it has not typically been historians who have examined the history of immunology.²⁰ Immunologists, much like psychiatrists and other physicians, have been interested in constructing the history of their field, some of them becoming professional historians themselves. Moreover, as Thomas Söderqvist, Craig Stillwell and Mark Jackson suggest, even the more critical work on immunology has not been particularly historical, but philosophical, in the case of Donna Haraway, Alfred Tauber and Pauline Mazumdar, and biographical, in the case of Thomas Söderqvist's biography of immunologist Niels Jerne.²¹ The task of situating the history of immunology into its various social contexts remains to be done, although the increasingly high profile of immune system diseases, most prominently AIDS, but also autoimmune diseases such as systemic lupus erythematosus, multiple sclerosis and rheumatoid arthritis, suggests that there will be many opportunities to do so.

The history of psychiatry can be said to have had the opposite problem, being at times so contextualised, theorised and politicised as to lose sight of the subject altogether. Roy Porter and Mark Micale state that 'in no branch of the history of science or medicine has there been less interpretive consensus' than in the history of psychiatry.²² They situate the roots of this divisiveness within psychiatry itself and its long history of competing mentalist and somatic philosophies.²³ Perhaps Andrea Tone's suggestion that during the last fifty years psychiatry

several of the participants ... that the exchange [between immunologists and historians] was meant to be unidirectional: scientists engaged in the production of immunological knowledge were there to tell historians "how things had really been". The resulting tension nearly turned into open confrontation between scientists and historians. In reviewing this episode the authors recalled Canguilhem's observation that the object of the history of science is different than the object of science. Alberto Cambrosio, Peter Keating, and Alfred I. Tauber, 'Introduction: Immunology as a Historical Object', *Journal of the History of Biology* 27 (1994), 375-8;

²⁰ When immunology has been studied by historians, issues related to vaccination and how scientists have conceptualised and described viral and bacteriological pathogens, rather than instances of immune system dysfunction, have tended to dominate. For example, in *Crafting Immunity*, a 2008 volume on the history of clinical immunology, five of the twelve chapters deal with vaccination and three others address the development of technologies used to detect viruses. Kenton Kroker, Pauline Mazumdar and Jennifer Keelan (eds), *Crafting Immunity: Working Histories of Clinical Immunology* (Aldershot: Ashgate, 2008).

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and history have taken divergent epistemological paths, towards and away from positivism, standardisation and objectivity respectively, also helps to describe the lack of consensus seen by Porter and Micale.²⁴ Just as psychiatry has experienced a wide variety of different theoretical paradigms, ranging from neurologically-based biological psychiatry to dynamically-oriented psychoanalysis, historians of psychiatry have embraced Whiggish, anti-psychiatric, new revisionist, Marxist, libertarian, feminist, hagiographic, intellectual, cultural, patient-centred and post-structuralist methodologies.²⁵ While these divisions have prevented the emergence of a clear and coherent depiction of psychiatry's history, they have resulted in a compelling historiography that tells as much about the practice of history, as it does about the history of psychiatry. Regardless, one of the challenges for the contemporary historian of psychiatry, operating in what Porter and Micale describe as the 'new "postideological" age', is coming to terms with these diverse and divisive methodologies and constructing one that suits the subject at hand and one's own interpretation of it.²⁶

Gauging where to situate the history of the Feingold diet amongst so many approaches is particularly important given the controversy that surrounded the diet and the numbers of different players involved in the story. One could take a Marxist anti-psychiatry approach, for example, and claim that the psychiatrists who 'tested' the Feingold diet were merely pawns of the pharmaceutical and food chemical industries which viewed the diet as a threat to the enormous profits made from selling hyperactivity drugs such as Ritalin and food additives themselves. Since hyperactivity was, and continues to be, the most commonly diagnosed child psychiatric condition, such an approach might also lead to the conclusion that psychiatrists were disinclined to support Feingold's theory since it transferred the aetiology of hyperactivity away from neurology and psychology of the mind and towards the

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field of allergy and immunology, thus reducing their ability to benefit financially from treating hyperactive children. Such a methodology would undoubtedly produce an entertaining story, but it would likely oversimplify how decisions about mental health treatment were made and underestimate the agency of hyperactive children, their parents and their physicians.

Similarly, a history of the Feingold diet that ignored the social, economic, professional and cultural contexts in which the diet was situated would overlook many of the most important elements of the story. Not only did the Feingold diet bridge allergy and psychiatry, but it also involved nutrition, education, parenting and notions about ‘the inherent perils of civilisation’.²⁷ The historian’s own views on these politically-laden issues will no doubt permeate any interpretation of the Feingold diet’s history no matter how diligent one is in disguising or downplaying them. Indeed, the histories of immunology that Anderson, Jackson and Rosenkrantz critique are not written from an Archimedean point; rather they betray a positivistic, deterministic faith in medical science’s ability to solve nature’s mysteries in an objective, logical manner.²⁸ In this way the histories of allergy/immunology and psychiatry are not as disparate as one might think. The responsibility of historians, given the inevitability of subjectivity, is to be self-aware and admit the interpretative nature of their inquiry.²⁹

Juxtaposing medical histories that are socially saturated against those that are lacking in contextual awareness belies the fact that there are excellent models to be found in both the histories of allergy and psychiatry that point to how a compromise between these approaches might be struck. Furthermore, such methodologies suggest that the dichotomy of

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biology/culture in the history of medicine is misleading with regards to how medical knowledge is developed, interpreted and applied. Micale, for example, has employed what he calls a ‘sociosomatic approach’ to the history of hysteria. This approach engages the historian in analyzing both the external (social) and internal (biological) aetiologies of disease and results in a pluralistic interpretation of mental illness. It seems as though such a flexible, multi-faceted approach, in which the weight of external and internal factors varies according to the subject, would also allow historians themselves to be pluralistic, rather than dogmatic when selecting potential methodologies to consider.³⁰

It could be said that Jackson takes a sociosomatic approach to his history of allergy. Jackson’s work is particularly instructive with respect to the history of the Feingold diet in that he has published on both the history of allergy and psychiatry. According to Jackson, the political and professional aspirations of mental health workers and allergists, respectively, contributed to expanding the concepts of feeble-mindedness and allergy.³¹ In *Allergy*, Jackson demonstrates how the meaning of allergy in the twentieth century has been shaped and reshaped not simply by clinical and laboratory science, but also by contemporary medical philosophies, such as the mid-century interest in psychosomatic medicine, ‘the global economy of allergy’, in particular the initiatives of the pharmaceutical, cleaning, cosmetics and food industries in making a profit from allergy, and anxieties about ‘the distinct health hazards supposedly intrinsic to modern Western lifestyles’.³²

Jackson’s depiction of allergist John Freeman’s emphasis on ‘the centrality of clinical experience (or the “experiential method”) over either theory or statistical evidence’ is especially pertinent to the history of the Feingold diet.³³ Like Feingold, Freeman trusted his

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clinical observations and was less concerned about the mechanisms underlying what he saw. Moreover, both clinicians believed in the individuality of their patients. For Freeman this meant having a ‘flexible approach to establishing therapeutic doses in particular patients’ and suggesting that clinicians ‘devised their own protocols and prepared their own allergen extracts to suit the demands of their own time and that of their patients’.³⁴ For his part, Feingold instructed parents employing his diet to become keen observers of their children’s behaviour, record their observations and make changes to the diet that reflected what they saw.³⁵ While this clinical, empirical approach satisfied both Freeman and Feingold, the reluctance of both to analyse thoroughly their theories and practices left them vulnerable to criticism.³⁶

Jackson’s willingness to delve into the laboratories, clinics and personal and professional lives of allergists such as Freeman (as well as Charles Richet, Clemens von Pirquet and others) and the larger political, economic, social and philosophical factors that influenced them, provides a solid framework for approaching Feingold and his work. Working in the history of immunology, Söderqvist and Tauber have also closely examined the lives of prominent immunologists, Niels Jerne and Elie Metchnikoff respectively, in order to understand not only the scientific origins of their theories, but the emotional and metaphysical roots as well. Söderqvist’s *Science as Autobiography* tackles the unconventional life of Jerne, ‘who would rather read Shakespeare and Proust than the *Journal of Immunology*’.³⁷ Söderqvist suggests that Jerne’s appreciation of Søren Kierkegaard’s philosophy, his elitist, vain, yet engaging personality and tumultuous emotional life influenced not only his professional style and scientific relationships, but the essence of his contributions to immunology, specifically selection theory of antibody

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formation and the network theory of the immune system. Stating that ‘in the summer of 1954 Jerne projected, metaphorically, entrenched patterns in his emotionally charged self-understanding and life experience to structure the puzzling experimental evidence he was confronted with in the laboratory’, Söderqvist explains how Jerne’s appreciation of absolute personal autonomy and ‘intellectual flexibility’ in human relations led him to conceptualise antibodies that were similarly autonomous and flexible in dealing with foreign molecules.³⁸ Söderqvist suggests that Jerne’s network theory also reflected the immunologist’s ideals when it came to his personal and professional relationships, lifestyle and personality.³⁹

It is common in the history of psychiatry, for example in the history of psychoanalysis, to search for the origins of psychiatric theory in the emotional life of the theorist; that Söderqvist uses a similar strategy in tracing the roots of post-war immunological theory suggests that immunology, despite its greater degree of scientific respectability, also has pronounced cultural underpinnings.⁴⁰ Söderqvist’s argument that science indeed equated to autobiography demands more evidence at times; for instance, more concrete examples of Jerne’s own ‘intellectual flexibility’ from the conversations he had with colleagues about culture and politics. Despite this shortcoming, Jerne’s own words, for example that ‘he wanted “to impress his personality like a stamp in colors and form; to be able to let nature reflect the depths of my own soul”’, do demonstrate how his immunological theories were an intended reflection of himself.⁴¹ Unfortunately, Feingold did not leave the extensive personal archive Jerne did, described as ‘tens of thousands of letters, notes, and manuscripts charmingly stored in hundreds of paper bags from the local supermarket’, but clues from his

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published work, letters and conversations help to uncover the emotional as well as the intellectual origins of Feingold's theory.⁴²

Although Tauber's work *The Immune Self: Theory or Metaphor?* is not a biography, as with Söderqvist's work on Jerne, it does contain insights into the philosophical underpinnings of early twentieth-century immunologist Elie Metchnikoff's holistic concept of self and corresponding phagocytosis theory. Tauber argues that Metchnikoff's thinking paralleled many contemporary philosophers, but especially Friedrich Nietzsche, whose philosophy he describes as being 'thoroughly permeated by, even tethered to, a biological self-consciousness'.⁴³ Nietzsche's concept of 'a self that is fundamentally and profoundly active, dialectical with its experience, and ever-changing and growing', is highly reminiscent of Metchnikoff's active, struggling, dynamic and thoroughly individualistic immunological self.⁴⁴ Such descriptions bear a similarity to Ilana Löwy's depiction of similar individualistic and holistic themes that arose in the contemporary research of Richet on anaphylaxis.⁴⁵ Although she suggests that Richet's experimental method was significant, using dogs, which Richet saw as having distinct personalities, as opposed to more homogeneous rodents, she also notes that Richet's diverse interests and monistic philosophy influenced his scientific theory.⁴⁶ Likewise, Tauber implies that the strangely similar psychological backgrounds of Nietzsche and Metchnikoff, both suffering from psychosomatic illness, severe manic-depression and nihilistic pessimism, help to explain the parallels between the philosopher and the scientist.⁴⁷

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Tauber's depiction of the philosophy underlying immunology and the concept of self is relevant to the history of the Feingold diet and psychiatric history in two significant ways. First, the manner in which Metchnikoff's theory of an active, individualistic, immunity conflicted with Paul Ehrlich's theory of passive, chemically-based, immunity is strikingly similar to twentieth-century debates about self between dynamic and biological psychiatrists.⁴⁸ Whereas dynamic psychiatrists shared a Metchnikovian emphasis on the mind's capacity to change and adapt to circumstance, leading them to treat patients with psychotherapy intended to foment positive change, biological psychiatrists favoured Ehrlich's 'image of a biochemically oriented science of organism reduced to the simplest physicochemical elements' in which the perceived neurological imbalances in patients' brains were treated with drugs.⁴⁹ In other words, psychiatrists have clearly participated in the twentieth-century 'struggle surrounding the ascendancy of reductionism, defining both the research strategies and the metaphysical foundations of biology and medicine'.⁵⁰ Feingold's theory complicated the simplistic, neurological understanding of hyperactivity that was emerging in the 1970s, but also undermined the psychiatric dichotomy of mind versus brain by claiming that, while hyperactivity was caused by food additives, it could also be exacerbated by emotional and environmental factors.⁵¹

Also relevant to the history of the Feingold diet is the manner in which the immunological community chose to ignore Metchnikoff's theory of immunity for a period of nearly fifty

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⁵⁰ Ibid., 44. On another level, however, biological and dynamic concepts of self converge in that they operate on an assumption that a patient's true self is one that is free of mental illness. But since societal norms strongly determine what is deemed to be mental illness, it could be argued that the true self that psychiatrists attempt to reveal is a social construction, rather than the actual essence of the individual. Diana Gittins' oral history of Severalls Hospital, for instance, describes many patients who enjoyed experiencing their 'altered states' and disliked when therapeutic measures reduced such opportunities. This is also relevant to hyperactive behaviour in children, which tends to be pathological only when it occurs in particular social settings, for example, in the classroom or at the dinner table as opposed to in the playground or on the football pitch. For psychiatrists, as for immunologists and philosophers, self is an elusive and problematic concept.

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years, from approximately 1908 (the year in which Ehrlich and Metchnikoff shared a Nobel Prize) to the late 1950s, a period called the ‘Dark Ages’ by historian of immunology Arthur Silverstein, only to be re-considered following the acceptance of Frank Macfarlane Burnet’s clonal selection theory.⁵² During this fifty-year period, chemists replaced biologists and physicians as the lead players in immunological research and their focus shifted from disease prevention and aetiology to uncovering the molecular mechanisms of immunity. Silverstein suggests that this shift was due to a number of factors, including a decline in the search for the pathogens that caused infectious diseases, the disruption of the First World War, which shifted the centre of the immunological world to the United States from Europe, and the death of Metchnikoff in 1916.⁵³ Finally, Tauber adds that the question of scientific legitimacy also played a role in resolving the debate: ‘reductionism was proposed as an attempt to better “objectify” the life sciences’.⁵⁴

The history of the Feingold diet, and the history of psychiatry generally, has been shaped by similar factors. As with Metchnikoff’s theory of immunity, Feingold’s theory was forgotten or ignored to a great extent after his death, although, as with the immunologist, a small number of followers continued to carry his ideas forward. Moreover, the respective professional styles and personality of each scientist seem to have dampened the resilience of their theories after their deaths. Metchnikoff, described by Paul de Kruif in *The Microbe Hunters* as ‘like some hysterical character out of one of Dostoevski’s novels’ was defensive about his theory and alienated many of his colleagues.⁵⁵ While Feingold’s personality was much more genial in public, he was fiercely defensive and protective of his theory, chose to circumvent scientific protocol by publishing his theory in a popular format and rejected suggestions that there was a need for his clinical observations to be supplemented by double-

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⁵⁵ Paul de Kruif quoted in

blind trials.⁵⁶ Just as the First World War disrupted the world of immunology, it could be that political factors, specifically a shift to the political right in North America and Britain during the 1980s, rendered some of the subversive, ecological elements of Feingold's theory less palatable to a more consumerist, conservative society.⁵⁷ Questions about the scientific legitimacy of psychiatry, like those mentioned by Tauber with respect to immunology, also influenced psychiatric acceptance of a theory that connected food additives and hyperactivity. Finally, as with Metchnikoff's theory, the Feingold diet, though still largely seen as an unorthodox approach to treating hyperactivity, has also re-emerged, particularly in Britain.

Although not social histories, the histories of immunology as presented by Söderqvist and Tauber reveal the role of many social factors in shaping how immunological knowledge evolved during the twentieth century. This suggests that the gap between the history of psychiatry, securely rooted in its social context, and that of immunology is not as great as one might think. The history of allergy reveals that there are even more tangible links between the immune system and the mind that help to situate Feingold's linkage of hyperactivity and food additives. Jackson, for example, observes that psychodynamic theories and treatment of allergy and asthma were prevalent during the post-war period. Such approaches 'served not only to consolidate radical liberal critiques of biomedical reductionism but also, paradoxically, to reinforce both contemporary fantasies of the good mother and reactionary pressures to condemn women to the domestic sphere in the aftermath of the Second World War'.⁵⁸ Carla Keirns has also recognised this association between emotions and allergy, describing how emotionally overbearing parents were blamed for causing asthma and hay fever in their children, although sibling rivalry and overcrowding were also seen as triggers.⁵⁹

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⁵⁷ The re-kindling of the Cold War during the 1980s, for example, shifted the focus of the environmental movement from the insidious effect of chemicals in the environment and in the food supply to the more pressing concern about nuclear annihilation.

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The post-war perception that allergic disease was one of the ‘trials and tribulations of modern civilised existence’ is another theme of Jackson’s *Allergy*. Allergy became ‘a convenient metaphor for the diverse physical, psychological and social perils facing modern populations’. Environmental historians are also beginning to stress this theme in an attempt to bring the histories of health and the environment together. The call for this convergence is warranted, according to Gregg Mitman, Michelle Murphy and Christopher Sellers, by vivid late twentieth-century examples of how environmental catastrophes are inseparable from their health implications.⁶⁰ Work by Mitman on the relationship between allergy and ecology, Sellers on contrasting water fluoridation/de-fluoridation strategies in the United States and India, Linda Nash on the insalubrious effect pesticides have had on migrant agricultural labourers in California and Harold Platt on the environmental struggles of Manchester and Chicago suggest that there are plenty of historical examples that demonstrate how health and the environment are inextricably linked.⁶¹

In particular, Mitman’s *Breathing Space* stresses how responses to commonly-targeted allergens such as ‘the stresses of civilization, pollens, cockroaches, air pollutants, molds, and dust mites’ in the twentieth and twenty-first centuries have ‘exacerbated the allergic landscape and made worse the very symptoms that we have aimed to relieve’.⁶² Although allergy sufferers fled to health resorts such as Denver, Colorado and Tucson, Arizona during the late nineteenth and early twentieth centuries for their clear skies and clean air, as early as the 1950s both cities had developed air pollution problems. In the case of Denver, its geography created an ideal situation for frequent temperature inversions, which ‘resulted in

⁶⁰ Mark Jackson is more circumspect, however, in linking epidemiological rises in allergy to environmental factors. For Jackson, ecological theories of allergy must be treated in the same critical fashion as any other aspect of allergy knowledge. Jackson, *Allergy*, 179-80.

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carbon monoxide and photochemical ozone problems equal to or greater than those in much larger metropolitan areas like Los Angeles and New York City. Denver became as famous for its “brown cloud” as it had once been for its “clear skies” and “fresh mountain air”.⁶³

Tucson’s air pollution was largely due to rapid urbanisation and resulting car fumes, but its pollen count, a measure of enormous importance to hay fever sufferers, had also ballooned due to the importation of non-native Bermuda grass and shade trees, intended to give the desert city the “civilized” look of eastern cities’.⁶⁴ As Mitman describes:

the former ecological haven had become an ecological hell. In a little more than twenty years, the atmospheric pollen load of allergenic plant species in Tucson had increased tenfold. Not only was the incidence of asthma now twice the national average, but also the incidence of hay fever was six to nine times greater.⁶⁵

Despite such ecological and health problems, Denver and Tucson continued to benefit economically from the allergic and asthmatic; both cities are currently centres of biomedical allergy research. But, as Mitman argues, it has only been wealthy Americans, those able to afford the move to such resorts in the past and with the means to pay for expensive allergy treatment today, who have benefited.⁶⁶ Poor inner-city minorities, whose rates of asthma and allergy are disproportionately higher than those living in affluent suburbs, have not.⁶⁷ As such, Mitman contends that the rise of allergy in the United States can only be mediated by understanding and taking steps to address its social and environmental causes.⁶⁸

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⁶⁴ The environmental devastation of ‘health communities’ Denver and Tucson provides an interesting comparison to the struggles faced by the civic leaders of ‘shock cities’ Manchester and Chicago in reversing decades of environmental neglect. While unbridled economic growth in all four cities resulted in dire ecological consequences, the environmental reformation of Manchester and Chicago described by Platt has also been spurred by economic motives.

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The degree to which changes to the natural environment, as well as the constructed environment, have affected health has also been raised by Peter Radetsky in his provocative *Allergic to the Twentieth Century*. Radetsky's sympathetic approach to the history of multiple chemical sensitivity (MCS), the reality of which is highly contentious, might resemble a conspiracy theory at times, but it nevertheless illustrates that in the last half-century, thousands of Americans have become convinced that the modern trappings of life are hazardous to health.⁶⁹ Such perceptions help to construct the context in which the Feingold diet became popular. On the other hand, the struggles that clinical ecologists such as the 'praised and reviled' Theron Randolph (1906-1995) had in establishing the legitimacy of his field and MCS helps to explain some of Feingold's difficulties in convincing the medical mainstream that his theory was credible.⁷⁰

Michelle Murphy's exploration of MCS also highlights the difficulties both physicians, such as Randolph, and MCS sufferers have experienced convincing 'the official mouthpieces of biomedicine that MCS is a legitimate diagnosis'.⁷¹ According to Murphy, MCS is an 'abjected' illness, an illness rendered into an impossibility by biomedicine and, thus, utterly rejected by mainstream physicians.⁷² Murphy emphasises that the history of MCS provides a challenge for the social historian, specifically because 'the tools of social constructivism and cultural studies will not always perform in the interest of those with whom my political sympathies lie'.⁷³ In other words, a typically constructivist approach might question the reality of MCS because the syndrome involves a multitude of symptoms and causes and, thus, appears to be particularly rooted in social, political and ideological factors. In order to overcome this difficulty, Murphy employs social constructivism in a 'relentlessly materialist'

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manner which acknowledges the physical and mental discomforts of MCS sufferers and allows her to achieve her stated goal of reaffirming the reality of MCS.⁷⁴ Murphy's conceptualisation of MCS is relevant for the history of the Feingold diet; while it is an appropriate historical task to deconstruct hyperactivity and reveal its numerous social contingencies, that task is separate from that of unravelling the web of factors involved in the assessment and reception of Feingold's theory.

Steve Kroll-Smith and H. Hugh Floyd also investigate the history of MCS, but their patient-centred approach focuses on exploring how those afflicted struggle to develop their own understanding of their affliction, or, in their words, to 'construct biomedical accounts of etiologies, pathophysiologies, and treatment regimens to explain and manage their debilitating and psychological symptoms'.⁷⁵ MCS, much like Feingold's hyperactivity, did not fit into a typical medical treatment model of drugs or therapy, nor did it fit into the presiding endogenous model of aetiology, whereby the root of illness was seen to exist within individual bodies and in genetic predisposition, rather than in the external environment.⁷⁶ Since MCS sufferers, as with Feingold adherents, have not been able to reform the capitalist system responsible for generating toxic environments and consumer products or the medical system which, according to the authors, works in the interest of capital production, they have instead abandoned these systems and become their own experts.⁷⁷ Kroll-Smith and Floyd suggest, however, that by demonstrating that the modernist separation of expert and lay

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⁷⁶ Kroll-Smith and Floyd acknowledge, as do Charles E. Rosenberg and William Rothstein, that exogenous theories of aetiology dominated in the nineteenth and early twentieth centuries when infectious diseases were more common and deadly. Nevertheless, as Sheila M. Rothman has shown with respect to tuberculosis, the morality and temperament of patients was also often blamed. Sheila M. Rothman, *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in America* (Baltimore: Johns Hopkins University Press, 1995);

⁷⁷ Paul Blanc also describes how the environmentally ill are often at the mercy of not only brazenly indiscriminate polluters, but 'run of the mill' manufacturers whose businesses are rarely constrained by environmental concerns.

knowledge in medicine is no longer tenable and by showing that capitalism can, or perhaps must, co-exist with environmental sustainability, reform of these systems is possible.⁷⁸

Kroll-Smith and Floyd's description of the politics behind expert systems and treatment alternatives has resonance for historians of psychiatry. The generation of expert knowledge in psychiatry, especially with regards to treatment, has been a contested enterprise in which 'experts' and their knowledge are often discarded when their explanations for and treatment of mental illness become either scientifically, economically, politically, or culturally untenable. In his exploration of the history of psycho-surgery, Jack Pressman uses the analogy of a lock and key to describe the relationship between perceived psychiatric problem and acceptable cure, the lock representing the problem that psychiatrists are meant to solve and the key being the therapeutic measure used to solve it. Since, as Pressman argues, psychiatric problems are culturally, as well as biologically, relative, they change as society does. When this happens, therefore, a different key, or therapeutic measure, is needed to address the changed lock, or problem.⁷⁹ This analogy is useful in addressing the reception of the Feingold diet within both medical and social contexts.

Erika Dyck uses a similar methodology in her investigation of the rise and fall of lysergic acid diethylamide (LSD) treatment for mental illness, especially alcoholism, during the 1950s and 1960s. According to Dyck, promising research findings and public support, particularly in the Canadian province of Saskatchewan where many of the most comprehensive studies were done, meant little during the mid-1960s when the media began blaming LSD addiction for crime and the American Food and Drug Administration (FDA) placed the drug on its list of illegal narcotics.⁸⁰ Another key factor was that Humphrey Osmond, the lead researcher in

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⁸⁰ 'Hitting Highs at Rock Bottom: LSD Treatment for Alcoholism, 1950-1970', *Social History of Medicine* 19 (2006), 313-29; Erika Dyck, *Psychedelic Psychiatry: LSD from Campus to Clinic* (Baltimore: The Johns

the Saskatchewan studies, exhibited a marked disdain for submitting his theories to double-blind controlled trials, arguing that such controls limited the experiences subjects might have while under the influence of LSD and, therefore, reducing the possible benefit and skewing the results of the trial.⁸¹ Osmond's criticism is strikingly similar to that of Feingold, who also argued that such trials were usually poorly, if not maliciously, designed to produce certain results and excessively delayed the potentially beneficial results of clinical observations for years. The potentials of both LSD and the Feingold diet as treatments for mental illness, therefore, were ultimately driven by factors both within and outside of the medical community.

LSD might have failed to become an accepted treatment for mental illness, but other psychoactive drugs, such as Miltown during the 1950s and 1960s, Valium during the 1970s and Prozac during the 1990s, would dominate psychiatric research and treatment by the 1970s. Although psychiatrist Jonathan Michel Metzler has challenged 'the notion that biological psychiatry replaced psychoanalysis', questioning the 'binary that ... academic psychiatrists, anthropologists, and historians of psychopharmacology assume exist between these two modes of treatment', his argument says more about the resilience of Freudian theory and gender stereotypes in American culture than it does about how the majority of psychiatrists have understood mental illness and treated patients during the past four decades.⁸² While Metzler's argument that prescribing patterns reinforced gender roles and that Freudian imagery was used in advertisements for psychiatric drugs is credible, it is difficult, given the power of pharmaceutical companies, the plethora of psychopharmacological research and 'the new biological language in psychiatry', to accept his claim that

Hopkins University Press, 2008), 128-32.

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⁸² Jonathan Michel Metzler, *Prozac on the Couch: Prescribing Gender in the Era of Wonder Drugs* (Durham, NC: Duke University Press, 2003), 4.

psychoanalytic theory remained the most potent force within American psychiatry following the 1970s.⁸³

If the history of hyperactivity is any indication, childhood mental illness during the 1960s and 1970s was increasingly conceived of as a neurological problem and treated using pharmacological means. Ritalin was first prescribed for children in 1961 and quickly became the most common way in which to treat the disorder ever since. Parents who disliked the idea of giving their children an amphetamine to treat their behavioural problems did not tend to turn to psychoanalytic explanations, but instead considered alternative biological explanations, such as Feingold's hypothesis. While Ritalin, as well as Valium, Prozac and other drugs, have generated considerable debate, they nevertheless became the mainstay of American psychiatric treatment during the last four decades of the twentieth century. The history of why, in the face of such controversy, these drugs have become so predominant helps to explain why other treatments, such as the Feingold diet and psychotherapy, have struggled to become or remain viable options.

Although Nicolas Rasmussen has suggested that psychoactive drugs can be seen as an extension of 'age-old human fantasies of magical cures and elixirs of youth', other historians have argued that the success of many drugs has also been carefully cultivated by pharmaceutical companies.⁸⁴ Historian David Herzberg, for example, contends that the popularity of tranquilisers and antidepressants was due in large part to changes in how pharmaceutical products were marketed by powerful pharmaceutical companies. Psychiatric drugs were advertised directly to physicians in medical journals during the 1950s and to patients via television commercials during the 1990s. These advertisements made

⁸³ Healy, *The Antidepressant Era*, 5.

⁸⁴ Nicolas Rasmussen, *On Speed: The Many Lives of Amphetamine* (New York: New York University Press, 2008), 2.

‘consumers out of doctors and patients’ and made medicine ‘part of a new consumerist “American dream” that reconfigured conceptions of what a good middle-class life – what happiness itself – ought to be like’.⁸⁵

Not all Americans, however, agreed with such definitions of contentment or middle-class life. As with the controversy that surrounded the numbers of children prescribed Ritalin during the 1970s, reaction to the increasing number of women being prescribed Valium during the same period indicate how arguments about psychiatric treatments mirrored broader societal debates. Feminist critiques of Valium, for example, not only addressed the drug’s addictiveness and side effects, but also called into question what the high prescription rates implied about women’s place in American society.⁸⁶ In other words, drugs such as Valium and Ritalin could ‘emblemize the nation’s systemic problems rather than offering a respite for them.’⁸⁷ Whereas the ‘Valium panic’ of the late 1970s resulted in a decrease in the drug’s popularity during the 1980s, dissatisfaction with Ritalin enticed many parents to turn to the Feingold diet.

Psychiatrist David Healy reiterates Herzberg’s emphasis on the role of advertising in raising the profile of psychiatric drugs, but goes further, claiming that pharmaceutical companies not only marketed antidepressants, but also the idea of depression.⁸⁸ As Healy maintains ‘In many respects the discovery of antidepressants has been the invention of and marketing of depression.’⁸⁹ Healy also contends that pharmaceutical companies worked with psychiatric

⁸⁵ Herzberg, *Happy Pills*, 4.

⁸⁶ Ali Haggett has argued that, despite the views of feminists such as Betty Frieden that the anxiety and depression experienced by women were due to their dissatisfaction with domestic life, many homemakers during the post-war period ‘considered their experiences to be both positive and worthwhile’. Ali Haggett, ‘Housewives, Neuroses, and the Domestic Environment in Britain, 1945-70’ in Mark Jackson (ed.), *Health and the Modern Home* (New York: Routledge, 2007), 84-110, at p. 85.

⁸⁷ Herzberg, *Happy Pills*, 149.

⁸⁸ Healy, *The Antidepressant Era*, 180-1; David Healy, *Let Them Eat Prozac: The Unhealthy Relationship Between the Pharmaceutical Industry and Depression* (New York: New York University Press, 2004), xii, 1-5.

⁸⁹ Healy, *The Antidepressant Era*, 5. Healy’s own relationship with the pharmaceutical industry might explain some of his attitudes toward it. In 2001 Healy was ready to begin a professorship at the University of

associations in Britain and the United States ‘to sell the idea that depression is underecognized in primary care and that it should be recognized and treated appropriately.’⁹⁰ Similar arguments were made by Schrag and Divoky during the 1970s with reference to hyperactivity and Ritalin and have been extended more recently by Peter Conrad and Deborah Potter with respect to how adults have increasingly been diagnosed with hyperactivity.⁹¹

Although it is clear that pharmaceutical companies and psychiatrists played a substantial role in promoting certain mental illnesses, viewing the emergence of depression and hyperactivity as a purely top-down phenomenon is problematic in that it ignores the role patients and parents play in not only accepting, but also advocating for, diagnoses to explain their or their children’s difficulties in coping with work, school and relationships. Anthropologist Allan Young, for example, has demonstrated how Vietnam War veterans lobbied successfully for post-traumatic stress disorder (PTSD) to be recognised as a legitimate mental illness during the 1970s.⁹² Hyperactivity advocacy groups such as Children and Adults with Attention Deficit/Hyperactivity Disorder (CHADD) have also played a considerable role in not only promoting hyperactivity as a legitimate disorder but also normalising the use of psychoactive drugs in its treatment.⁹³ The viability of new medical ideas, therefore, has often been based on the creation of symbiotic networks between physicians, patients, industry and government.

Toronto, but his job offer was revoked after he made a speech in which he claimed that Prozac, made by Eli Lilly, could cause patients to become homicidal or suicidal. Although the University of Toronto denied that the speech contributed to the revocation of Healy’s job offer, Eli Lilly was a major sponsor of the University’s Centre for Addictions and Mental Health and had a history of collaboration with the University dating back to the first trials of insulin to treat diabetes during the early 1920s. Anonymous, ‘Professor Says He Lost Job After Criticising Prozac’, 18 April 2001, www.cbc.ca/canada/story/2001/04/17/healy010417.html, accessed 9 May 2009.

⁹⁰ Healy, *The Antidepressant Era*, 181.

⁹¹ Schrag and Divoky, *The Myth of the Hyperactive Child*, 80-115; Peter Conrad and Deborah Potter, ‘From Hyperactive Children to ADHD Adults: Observations on the Expansion of Medical Categories’, *Social Problems* 47 (2000), 559-82.

⁹² Allan Young, *The Harmony of Illusions: Inventing Posttraumatic Stress Disorder* (Princeton: Princeton University Press, 1995), 108-42.

⁹³ CHADD, ‘Understanding AD/HD’, www.chadd.org, accessed 9 May 2009.

This process of authorising medical knowledge by building new or exploiting established medical networks has been continuously explored by historians, including Rima Apple and Harmke Kamminga with reference to the discovery of vitamins and Bruno Latour and Peter Atkins on the rise of pasteurisation in France and England.⁹⁴

That many families were able to employ the Feingold diet successfully, despite the disapproval of most physicians, not only demonstrates the importance of non-scientific networks in the dissemination and reception of medical ideas, it also hints at the agency employed by parents in determining their children's medical treatment. This reflects Rima Apple's suggestion that mothers during the late twentieth century increasingly questioned medical authority when it came to advice on how to raise their children.⁹⁵ Apple's research also reminds the historian, however, that American mothers came from a wide range of social, educational and ethnic backgrounds, and that it is misleading to generalise their experiences. In order to avoid this danger, oral history has been employed to explore parents' and patients' experiences of the Feingold diet and to determine what patterns can be detected regarding the families who were able to employ the diet successfully. Although some trends did emerge, it also became clear that, as a whole, Feingold families were socioeconomically, ideologically and ethnically different. The oral history also indicated that Feingold families were more able to adhere to the diet than medical researchers assumed. By questioning assumptions made by physicians about the experiences of patients and their families, historians can develop more sophisticated understandings of mental illness and the mentally ill. Ali Haggett, for example, has used oral history to question many of the assumptions

⁹⁴ Bruno Latour, *The Pasteurization of France*, trans. by Alan Sheridan and John Law (London: Harvard University Press, 1988);

⁹⁵ Rima Apple, *Perfect Motherhood: Science and Childrearing in America* (New Brunswick, New Jersey: Rutgers University Press, 2006), 135-53

made by historians about how British women during the post-war period felt about their role as mothers and homemakers and has also deconstructed why these assumptions were made.⁹⁶

There is a risk that when discussing families' reactions to the Feingold diet, the views of children may be overlooked. Historians such as Harry Hendrick have stressed that many so-called histories of children are instead histories of the concept of childhood as understood by adults and, as such, are not particularly 'child-centred'.⁹⁷ In Hendrick's work, however, the ability of children to affect change or influence decision-making, even in subtle ways, is quite limited; moreover, children are not seen by policy makers as ends in themselves, but rather 'they are to be possessed in order to maximise their potential as investments in *our* future'.⁹⁸ When one examines sociological and historical research on the history of hyperactivity, Hendrick's model and concerns are appropriate. Most of the studies conducted by sociologists such as Schrag and Divoky, Conrad, Ilina Singh, Adam Rafalovich and others, for example, have been top-down, portraying children as hapless victims of their diagnosis, and concentrating instead on the actions and experiences of parents, physicians and teachers.⁹⁹

With regards to the history of the Feingold diet, however, the agency of children is inescapable and, therefore, it is important to include a strong, if slightly altered, element of E. P. Thompson's notion of 'history from below'. Much of the impetus to produce histories which are grounded in the experiences, opinions and narratives of everyday people originated in 1963 when Thompson published *The Making of the English Working Class*. Thompson

⁹⁶ Haggett argues that the perception that British housewives were unhappy was created in part because 'contemporary feminist social theorists and feminist historians originated from untypical backgrounds that were either highly academic or political, and thus were largely unrepresentative of the average suburban housewife'. Haggett, 'Housewives', 84-5, 104-5.

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⁹⁸ Italics in original.

⁹⁹ Schrag and Divoky, *The Myth of the Hyperactive Child*;

sought ‘to rescue the poor stockinger, the Luddite cropper, the “obsolete” hand-loom weaver, the “Utopian” artisan, and even the deluded follower of Joanna Southcott, from the enormous condescension of posterity’.¹⁰⁰ Thompson’s call for ‘history from below’ has been echoed with respect to the history of child and adolescent health by historians such as Hendrick, but also Roger Cooter, Dorothy Atkinson, Mark Jackson and Jan Walmsley, and Diana Gittins.¹⁰¹ Telling the stories of the mad, infirm, unclean, unhealthy and disordered, though important and laudable, is not enough; such accounts must be integrated into those of health professionals, policy makers and medical researchers, as well as into the relevant societal context, in order to develop more comprehensive and instructive histories of medicine. Fortunately, there have been histories of child and adolescent health that have taken steps towards this goal, for example those written by Heather Munro Prescott, Katherine W. Jones, Cynthia Comacchio and Gemma Blok on adolescent medicine in the United States, American child guidance clinics, adolescent health in English Canada and Dutch therapeutic communities for disturbed youth, respectively.¹⁰²

Can the history of the Feingold diet exist as both a history of psychiatry and allergy? The broader historiographies of these two subjects suggest that the barriers between medical fields, as well as the scientific enterprises that inform them, are somewhat artificial. In a direct sense, while allergies have been thought to have been caused or exacerbated by psychological disturbance and judged to be psychosomatic, the Feingold diet represented one of a number of theories which posited that mental health problems could be rooted in allergy. In another way, the histories of allergy and psychiatry also demonstrate that people afflicted with mental illness and allergy both suffer from adverse reactions to what is for them a

¹⁰⁰ E. P. Thompson, *The Making of the English Working Class* (London: Gollancz, 1963), 12-13.

¹⁰¹ Roger Cooter, ‘Introduction’, in Roger Cooter (ed.), *In the Name of the Child: Health and Welfare, 1880-1940* (London: Routledge, 1992), 1-18, at p. 9;

¹⁰² Heather Munro Prescott, *A Doctor of Their Own: The History of Adolescent Medicine* (Cambridge, Massachusetts: Harvard University Press, 1998);

hostile environment. In other words, their immunological or emotional self is at odds with the physical or social stimuli to which they are exposed. For the allergic, the physical environment of foods, animals, dust, plants and countless synthetic materials provide challenges with which their body, for quixotic, impractical reasons or, possibly, for reasons we do not yet understand, struggles to cope.

For the mentally ill, it is often a hostile social environment that does not accommodate to their socially unacceptable behaviour. Both categories of sufferers, in most cases, lack the political power to change these environments, and subsequently look to medicine to adjust their immunological or emotional self. When medicine has been found wanting, both the allergic and the mentally ill have looked for alternative solutions, as have those clinicians who have become similarly frustrated by the ineffectiveness of modern allergic medicine or psychiatry. Feingold's approach to hyperactivity and allergy brought both concepts together, thus shattering the modernist dichotomies between mind and body, expert and lay, biology and culture and civilisation and nature. Therefore, the history of the Feingold diet becomes not only a history of psychiatry or allergy, but a chapter in the history of how humans respond, somatically, emotionally and metaphorically, to the civilisations they create.

Part I

The Origins of the Feingold Diet

Chapter 2

The First Hyperactive Children

The history of the Feingold diet is a key episode in the history of hyperactivity, a disorder that has been seldom examined by historians. Moreover, the contentious and confusing nature of hyperactivity helps to explain both the popularity and the denunciation of the Feingold diet. In order to understand the history of the Feingold diet, it is important to comprehend the broader history of hyperactivity and, specifically, the context in which the notion of hyperactivity emerged.¹⁰³ Unfortunately, many accounts of the history of hyperactivity have failed to recognise its social underpinnings. This makes it difficult to understand why hyperactivity became such an important and controversial topic to American parents, physicians, teachers and politicians during the 1960s, and also why an alternative approach to the disorder, the Feingold diet, became so appealing and so divisive.

Despite the fact that hyperactivity was rarely discussed in either medical or educational circles until the mid 1950s, many medical texts, self-help manuals aimed at parents and even the handful of historical work focusing on the disorder have suggested that the history of hyperactivity dates back to at least the mid-nineteenth-century, and that it can be diagnosed retrospectively in historical figures such as Lord Byron, Wolfgang Amadeus Mozart, Oliver Cromwell and Winston Churchill.¹⁰⁴ In some ways this early history of hyperactivity can be

¹⁰³ Inquiries into the context into which hyperactivity emerged might also shed light on why the disorder became a commonly diagnosed childhood psychiatric condition in the United States well before anywhere else, particularly Britain. Although Steven Box's preface to British edition of Schrag and Divoky's *The Myth of the Hyperactive Child* provides some insight into why this has been the case, Britain's hesitation to diagnose children with hyperactivity is also discussed in Chapter 11. Steven Box, 'Preface', in Peter Schrag and Diane Divoky, *The Myth of the Hyperactive Child: And Other Means of Child Control* (New York: Penguin Books, [1975] 1982), 7-30.

¹⁰⁴ For example: Paul H. Wender, *ADHD: Attention-Deficit Hyperactivity Disorder in Children and Adults* (Oxford: Oxford University Press, 2000), 3; Historical and sociological works which suggest that hyperactivity has a history dating back to the nineteenth and early twentieth centuries include:

seen as the ‘prehistory’ of hyperactivity. In comparison to the considerable public profile hyperactivity has held during the last half century, the prehistory of hyperactivity is characterised by sporadic medical interest and limited, if any, public awareness of the disorder.¹⁰⁵ While the late twentieth century can include Dennis the Menace, Calvin (from the comic strip *Calvin and Hobbes*) and Bart Simpson as icons of hyperactivity, and the medical and popular literature has produced thousands of studies and stories about hyperactivity, hyperactive children during the first half of the twentieth century did not attract medical or cultural attention.

The prehistory of hyperactivity demonstrates that hyperactive, impulsive and inattentive behaviour has long been associated with other types of problematic childhood behaviour. Although it was not until the 1950s that such behaviour was seen as intrinsically pathological, an analysis of the incidents in which similar behaviours have been identified nonetheless shows how psychiatrists and paediatricians began associating troubling childhood behaviour with neurological dysfunction as early as 1900. Analysing the studies identified as precursors to modern research into hyperactivity also helps to trace how, as beliefs about aetiology changed, so too did the labels used to describe such behaviour. These changes, in turn, affected how many children could be diagnosed as having a behavioural disorder such as hyperactivity.

Finally, the prehistory of hyperactivity is an example of how history can be used by interested parties to shape the understanding of a disorder. Writers who focus on the prehistory of

For post mortem diagnoses of hyperactivity in historical figures, see: Michael Fitzgerald, ‘Wolfgang Amadeus Mozart: The Allegro Composer’, *Canadian Journal of Diagnosis* 17 (2000), 61-4;

¹⁰⁵ This is not to suggest that awareness of particular medical conditions cannot fluctuate. Elizabeth Siegel Watkins has described how medical interest in male menopause emerged first during the late 1930s and continued to medical attention until the mid-1950s. The topic then disappeared from medical journals until the late 1990s, when the notion of male menopause was ‘repackaged as andropause’, and ‘what had been previously construed as a social problem – the travails of mid-life – was refashioned as a medical problem’, specifically ‘testosterone deficiency’. Elizabeth Siegel Watkins, ‘The Medicalisation of Male Menopause in America’, *Social History of Medicine* 20 (2007), 369-88, at p. 371.

hyperactivity do not do so because it is inherently more captivating than the more recent past; instead, they are engaged primarily in a rhetorical process of creating a more scientifically-based history for a disorder whose roots also delve substantially into the terrain of the social, cultural and political. As will be demonstrated in Chapter 3, Feingold used similar strategies to create a politically palatable history of his own theory of hyperactivity. Deconstructing the prehistory of hyperactivity, as well as Feingold's version of the origins of the Feingold diet, indicates not only that the history of disease may differ from one account to another, but also that history can be utilised to sculpt an image of a disorder that accords with an acceptable version of how medical science operates.

This chapter begins by examining the prehistory of hyperactivity and argues that early observations of so-called hyperactive behaviour by physicians were markedly different from descriptions of the disorder that emerged during the late 1950s. It then proceeds to explain how and why the modern notion of hyperactivity emerged during this period, suggesting that one of the key catalysts for the popularisation of the disorder in the United States was the launch of *Sputnik* by the Soviet Union and the growing perception that American children were academically inferior to their Soviet counterparts. The chapter concludes by demonstrating that, as hyperactivity became a commonly diagnosed disorder during the 1960s and 1970s, it also became a topic of intense debate.

The Prehistory of Hyperactivity

According to most accounts of the prehistory of hyperactivity, the first hyperactive child was Fidgety Philip, a character in a series of nursery rhymes written by German paediatrician, Heinrich Hoffman, and published in 1845. Fidgety Philip:

... won't sit still
He wriggles
And giggles

And then I declare,
 Swung backward and forward,
 And tilts up his chair
 Just like any rocking horse;-
 ‘Philip I am getting cross!’

See the naughty, restless child,
 Growing still more rude and wild,
 Till his chair falls over quite.
 Philip screams with all his might,
 Catches at the cloth, but then
 That makes matters worse again.
 Down upon the ground they fall,
 Glasses, bread, knives forks and all.
 How Mamma did fret and frown,
 When she saw them tumbling down!
 And Papa made such a face!
 Philip is in sad disgrace...¹⁰⁶

The fact that Hoffman was a paediatrician seems to have inspired writers who cite Hoffman’s poem as the first identification of hyperactive behaviour. Nevertheless, Hoffman’s intent in writing *Struwwelpeter*, which comprises nine other nursery rhymes, including ‘The Story of Little Suck-a-Thumb’ and ‘The Dreadful Story of Pauline and the Matches’, was not to describe pathological child behaviour. Dissatisfied with the quality of children’s literature, he wrote the poems to entertain his young son, Carl. As children’s literature expert Jack Zipes describes, ‘*Struwwelpeter* is a funny manual of good sense ... tell[ing] children, especially middle-class children, in graphic detail exactly what will happen to them if they do not do as they are told.’¹⁰⁷ Hoffman’s profession notwithstanding, there was nothing inherently pathological in Fidgety Philip’s behaviour, he was merely one of a number of naughty children depicted to show the (admittedly amusing and somewhat macabre) consequences of behaving badly.

¹⁰⁶ Heinrich Hoffman, *The Story of Fidgety Philip* (1845) www.fln.vcu.edu/struwwel/philipp_e.html, accessed 24 January 2009. More recently, *Struwwelpeter*, which translates into English as *Shockheaded Peter*, has inspired the Tiger Lilies, a London musical troupe, to create *Shockheaded Peter: A Junk Opera*.

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Similarly, the early accounts of pathological childhood behaviour cited in medical literature as precursors of hyperactivity often bear little in common with later descriptions of the disorder. Although most accounts of the early history of hyperactivity cite Still's speech to the Royal College of Physicians of London in 1902 as the first mention of behaviours resembling hyperactivity in a medical context, psychiatrists Seija Sandberg and Joanne Barton have referenced earlier observations of such behaviour, especially those by Thomas Clouston (1840-1915) in 1899. Clouston, lecturer at the University of Edinburgh and Physician Superintendent of the Royal Edinburgh Asylum, described three 'very difficult morbid conditions in neurotic children, conditions which lie on the borderland of psychiatry', specifically, 'simple hyper-excitability', 'hypersensitiveness' and 'mental explosiveness'.¹⁰⁸

It is perhaps ironic that Clouston is rarely mentioned in the prehistory of hyperactivity because his descriptions of these conditions are superficially more similar to depictions of hyperactivity that emerged during the 1960s than others written during the early twentieth century. His portrayal of the hyper-excitable child who 'becomes ceaselessly active, but ever-changing in its activity' and suffers from 'undue brain reactivity to mental and emotional stimuli', for example, neatly encapsulated the hyperactivity, impulsivity and distractibility that have typified hyperactive children for the last half-century.¹⁰⁹ Clouston's account of such behaviour, however, also bore key differences from those written later. First, he stated that hyper-excitable behaviour 'only lasts for perhaps a few months or a year', a key difference from later assessments which stressed that hyperactivity was a much more permanent condition, lasting until puberty, if not interminably. Second, he emphasised that mentally explosive children, those who were prone to irritable, impulsive, violent and defiant behaviour, were most often girls, not boys.¹¹⁰ This characteristic is opposite to later

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descriptions of the epidemiology of hyperactivity, which demonstrate that boys have been more frequently diagnosed with the disorder.¹¹¹ Despite these differences, Clouston's observations, not to mention his preference for using 'large doses' of bromides to treat such children ('to the point when the symptoms of brominism are beginning to show themselves'), and his belief that such conditions were rooted in the cerebral cortex, bear greater resemblance to today's understanding of hyperactivity and how to treat it than those that followed during the next fifty years, including the often-cited observations of Sir George Still.¹¹²

Sir George Still (1868-1941), whose speech was in fact the Cloustonian Lecture for 1902, named after Clouston, is best known generally for being one of Britain's first paediatricians and for describing Still's disease, a form of juvenile arthritis.¹¹³ For those interested in the history of hyperactivity, however, his fame comes from his description of 'children who show a temporary or permanent defect in moral control ... but pass for children of normal intellect and were not otherwise believed to be insane.'¹¹⁴ Still's aim in delivering the Cloustonian Lectures, which were subsequently published in the *Lancet*, was to address the question: 'Is diminution or defect of moral control in children ever the manifestation of a morbid mental state ... and if so, under which conditions does it occur?'¹¹⁵

In answering these questions Still insisted that such defects in moral control were pathological, and that the conditions under which such defects occurred were varied. Still's

¹¹¹ Many psychiatrists believe that girls have been under-diagnosed because they tend not to exhibit the 'explosive' behaviour that Clouston associated with them. Rather, they tend to be quiet, but inattentive and unable to focus.

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¹¹³ Still was described by a contemporary as 'a model of propriety', 'reserved', 'rigidly Victorian', 'conservative by nature'; 'he never told a funny story; he never wanted to hear one'. He was also "'abnormally reticent" except with small children [especially girls, whom he preferred to boys] between the ages of three and ten'. From Eric G. L. Bywaters, 'George Frederic Still (1868-1941): His Life and Work', *Journal of Medical Biography* 2 (1994), 125-31, at pp. 127-9.

¹¹⁴ Still, 'Cloustonian Lectures', 1008.

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definition of ‘moral control’ was far-reaching, encompassing ‘the control of action in conformity with the idea of the good of all [...and] the good ofself’.¹¹⁶ Defects in moral control resulted in a wide range of qualities including, in order of frequency, ‘passionateness [susceptibility to passion, intensity of emotion or anger]’, ‘spitefulness-cruelty’, ‘jealousy’, ‘lawlessness’, ‘dishonesty’, ‘wanton mischievousness-destructiveness’, ‘shamelessness-immodesty’, ‘sexual immorality’ and ‘viciousness’. Still believed that at the heart of these qualities was ‘the immediate gratification of self without regard either to the good of others or to the larger and more remote good ofself’.¹¹⁷ He added that the loss of moral control could either be an inherent dysfunction or an acquired dysfunction following a history of exhibiting moral control. This latter category, however, was distinct from acquired lack of moral control caused by illness or brain injury.

When the twenty cases Still described are analysed, however, profound differences emerge between them and the children diagnosed during the time of the Feingold diet.¹¹⁸ First, Still admitted that he had to make a ‘special effort to seek out’ the twenty children that made up his study. In Still’s words, such cases were ‘by no means common’.¹¹⁹ This is not surprising when the histories of the children he described are examined. Most of the twenty children appear to be significantly disturbed, capable of inflicting brutal violence on other children, their parents, animals and themselves, and many were either institutionalised or thought to be headed for such a fate.

Moreover, most of the specific behaviours Still described were either distinct from or not necessarily associated with hyperactivity today, including pica (eating inedible substances

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¹¹⁸ Rafolovich has also criticised the tendency to link what Still described to modern conceptions of hyperactivity.

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such as dirt or paper), extreme violence, self-harm, pathological dishonesty, sexual immorality and theft. Inattention and fidgety behaviour were mentioned, but they were not the core behaviours that Still described. In the case of Still, as well as that of Clouston to a lesser extent, hyperactivity was only one of a series of symptoms of underlying pathology; it was not a disorder in itself. Nevertheless, what both Still and Clouston did accomplish was to identify a small group of children who were neither intellectually disabled or brain damaged, but whose troubling behaviour was similar to children with such conditions. In so doing, they began the process of applying medical terminology and aetiology to socially and educationally inappropriate behaviours exhibited by children. It is more so this process, rather than the identification of hyperactivity in children, which bears a resemblance to the research conducted by child psychiatrists on hyperactivity half a century later.¹²⁰

Still's description of defective moral control in children, as well as Clouston's observations, reflected late Victorian concerns about behavioural and intellectual disability in children and, especially, children who were not so impaired that they would be routinely institutionalised. As Mark Jackson has described, individuals who occupied 'the borderland of imbecility' were believed to be a burden on society and a potential threat to social order.¹²¹ Both the children Still described and those diagnosed with hyperactivity decades later occupied such a borderland, a conceptual space, as Jackson puts it, 'ambiguously situated between the supposedly pathological and the normal'.¹²² Moreover, the desire to categorise such individuals 'appears to have been inspired not primarily by cognitive developments in science and medicine but by the administrative, educational, and medical problems generated by institutional expansion in the middle decades of the nineteenth century'.¹²³ The education

¹²⁰ Again, this argument expands somewhat on that made by Rafalovich.

¹²¹ Mark Jackson, *The Borderland of Imbecility: Medicine, Society and the Fabrication of the Feeble Mind in Late Victorian and Edwardian England* (Manchester: Manchester University Press, 2000), 1-5.

¹²² *Ibid.*, 12.

¹²³ *Ibid.*, 28.

legislation during the 1860s and 1870s, which required that more children attend school and exposed those who had difficulty learning, also contributed to the interest in and classification of marginal learners.¹²⁴ As will be discussed below and in Chapter 6, similar pressures on the education system, as well as changes to the provision of psychiatric care, also affected debates about hyperactivity during the 1960s.

The next instance of hyperactive behaviour commonly cited in medical accounts of the history of hyperactivity focuses on children suffering from post-encephalitic disorder during the early 1920s. *Encephalitis lethargica*, von Economo disease or sleeping sickness was a perplexing disorder that grew to epidemic proportions during the late-1910s, only to disappear during the late 1920s. While the disease resulted in a wide range of symptoms, including lethargy, fever, headache and catatonia, the residual effects of the disease, described as post-encephalitic disorder, were equally troubling, including physical impairments, eating and sleeping disorders and socially disruptive behaviour ranging from ‘excessive naughtiness to gross criminal acts’.¹²⁵ For instance, Franklin G. Ebaugh, Director of the Neuropsychiatric Department at the Philadelphia General Hospital, stated that sexual precocity was exhibited in two of the seventeen cases he saw, and that violent behaviour was evident in many others. While one ‘patient tried to kill other members of his family’, another ‘stabbed a schoolmate with a knife’.¹²⁶ Ebaugh also cited depression (including suicide attempts), hysteria, involuntary tics, insomnia, narcolepsy, dizziness, headaches, visual disturbance and mental deficiency as other common symptoms.¹²⁷

¹²⁴ Ibid., 25-7.

¹²⁵ As the title of the article indicates, post-encephalitic disorder was thought to be severe enough to warrant leucotomy (lobotomy) in some cases. In Thorpe’s paper, he emphasises that psychosurgery was an option due to the ‘gloomy’ prognosis in most cases. The two adult patients, who had nonetheless contracted encephalitis when they were children, were ‘content and happy’, yet ‘still irresponsible ... as judged by normal social standards’, following their prefrontal leucotomies.

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The wide range of symptoms included in post-encephalitic disorder was even more diverse than those described by Still, but the cause of the disorder echoed Still's description of 'morbid defect of moral control associated with physical disease'. Whereas Still associated such behaviour with diseases and injuries ranging from tumours and meningitis to blows to the head and acute rheumatism, the cause of post-encephalitic disorder was made clear by its name.¹²⁸ More important for the development of child psychiatry, however, was the association of abnormal behaviour with neurological trauma, resulting from infection, injury, auto-immune dysfunction or prenatal or postnatal respiratory problems.

Although this association led subsequent researchers, such as Eugen Kahn and Louis Cohen during the 1930s, and Alfred Strauss and Heinz Werner during the 1940s, to study this link further, with the latter pair eventually describing what they observed as 'minimal brain damage', it also helped to establish for many child psychiatrists that such behaviour was rooted in neurological, rather than psychological, dysfunction.¹²⁹ In the words of Dr Myerson, who commented on Kahn and Cohen's presentation to the Massachusetts Psychiatric Association in 1933: 'I think that encephalitis has probably illuminated the genesis of personality more than all the psychological work that has been done. I say this with all due respect to the psychologists who are here present.'¹³⁰ In other words, childhood behaviour disorder was a disease of the brain, and not the mind.

Another significant development in the prehistory of hyperactivity during the 1930s had implications for how the disorder would come to be commonly treated. At Emma Pendleton

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¹³⁰ Meyerson quoted in As discussed in detail in Chapter 4, the other physicians to recognise hyperactive behaviour were allergists, typically food allergists. In fact, there were as many, if not more, articles mentioning hyperactivity as a specific condition, separate from violent or dishonest behaviour, in allergy periodicals during the 1910s to 1940s, as there were in psychiatry journals. Although food allergists recognised this other aspect to the prehistory of hyperactivity, psychiatrists did not tend to do so.

Bradley Home, a children's psychiatric asylum in Rhode Island, the prevailing belief was that psychiatric problems were largely neurological in nature, and that neurosurgical remedies were often warranted.¹³¹ This reflected the beliefs of many American psychiatrists during the 1930s, since the psychoanalysts from Nazi Germany and elsewhere in central Europe, who would come to dominate post-war American psychiatry, had not yet begun their exodus to North America. Charles Bradley (1902-1979), a great nephew of the Home's founders, headed the medical staff and utilised pneumoencephalography in his neurological evaluation of patients. The painful procedure involved draining much of the cerebrospinal fluid from around the brain with a spinal tap and replacing it with oxygen, helium or air in order to improve x-ray images of the brain.¹³² Not unexpectedly, the operation resulted in severe headaches and nausea, and in 1937 Bradley prescribed the amphetamine Benzedrine in an effort to stimulate the replacement of spinal fluid and ease the children's headaches. Unfortunately, the stimulant did little for the headaches, but teachers at the Home observed that it seemed to improve the ability of patients to learn and behave at school.¹³³ After testing the drug further, Bradley began using it regularly and, by 1950, had used it on 275 children and found that it was effective over sixty per cent of the time.¹³⁴

Although *AJP* has described Bradley's discovery as one of 'the most important psychiatric treatment discoveries', the journal also recognised that although 'Bradley and his colleagues published their observations in prominent journals and they were reported in the media as well, 25 years passed before anyone attempted to replicate his observations, and more than 25 years passed before stimulants became widely used for ADHD'.¹³⁵ The quarter-century gap

¹³¹ This contrasts somewhat with sociologist Iina Singh's suggestion that the Bradley Home was 'grounded in a combination of behaviorist, psychoanalytic, and mental hygienist principles', although Singh does discuss Bradley's 'more active biomedical interventions'. Singh, 'Bad Boys, Good Mothers', 589.

¹³² In the 1973 horror film *The Exorcist*, the possessed child Regan McNeil, whose behaviour certainly resembles that of the children described by George Still, undergoes pneumoencephalography. By the 1980s, computed tomography (CT) scans had largely replaced the painful and dangerous procedure.

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between Bradley's discovery and its application by child psychiatrists highlights how context can impact upon the acceptance of new psychiatric conditions and treatments. Although Bradley's discovery came thirteen years before the synthesis of the first anti-psychotic, Chlorpromazine, in 1950, his observations nonetheless coincided with the emergence of other types of neurologically-based psychiatric treatment, such as insulin shock therapy (1933), electroconvulsive therapy (1934) and leucotomy or lobotomy (1935). Given the heroic, and inherently dangerous, nature of these treatments, it is difficult to imagine that psychiatrists would be averse to prescribing a stimulant, even to children.

The problem with Bradley's discovery, then, had less to do with the supply of an acceptable treatment than the demand for it. Bradley stumbled onto his findings trying to ease the headaches of children who had undergone a spinal tap; his observations that Benzedrine appeared to improve learning and behaviour were merely tangential. If there had been a greater demand for treatment alternatives for hyperactive children during the late 1930s, it is likely that Bradley's article in *AJP* would have created more of an immediate impact. The fact that it did not do so suggests that such children were not perceived to be of major psychiatric concern until much later, when Bradley's discovery was taken up with alacrity. Nevertheless, most medical accounts of the history of hyperactivity gloss over the twenty-five year gap and emphasise the long tradition of using stimulants to treat hyperactivity. Although such accounts are correct in the sense that Bradley's discovery did provide a reference point to which subsequent biological psychiatrists could refer, thus underlining the legitimacy of stimulant treatment of hyperactivity, there was no established tradition of prescribing stimulants to children with behaviour problems until the emergence of drugs such as Ritalin during the 1960s.

A number of similar problems arise when trying to connect investigations of children's behavioural disorders during the early part of the twentieth century to conceptions of hyperactivity which emerged during the 1950s. The first is that, as mentioned above, hyperactivity and inattention were only two of a wide range of behavioural problems identified in post-encephalitic disorder and minimal brain damage. They were also not as readily apparent or striking as other behaviours exhibited in these conditions, such as the extreme violence, criminal behaviour and self-harm. Children exhibiting such symptoms were also a rarity, owing, in part, to the relative infrequency of childhood diseases and injuries that could inflict such damage on the brain.

Furthermore, unlike the vast majority of cases of hyperactivity that would be diagnosed during the 1960s, the aetiology of post-encephalitic disorder and minimal brain damage was self-evident. Since brain damage was not evident in the histories of most patients, researchers investigating hyperactivity during the 1960s and onwards could only speculate about aetiology. Connection between the two constellations of behaviour, therefore, was fairly tenuous. Because of this aetiological disjunction, researchers during the 1960s had largely replaced the term 'minimal brain damage', which specified that those afflicted had suffered some form of brain injury, with the vaguer term 'minimal brain dysfunction', which included both brain damaged individuals and those whose neurological dysfunction was of unknown origin.¹³⁶ Despite these incongruities, most histories of hyperactivity found in medical books describe post-encephalitic disorder and minimal brain damage as precursors of hyperactivity.

If the links between early depictions of so-called hyperactivity emerging in Hoffman, Still and others, and modern conceptions of hyperactivity emerging during the 1950s and 1960s

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are so fragile, why are they included, indeed emphasised, in most accounts of the history of hyperactivity? The desire to extend the history of hyperactivity into past centuries appears to have been influenced by current mainstream medical opinion that hyperactivity is a genetic neurological disorder and, therefore, has always existed in the human population. Identifying instances of such behaviour in previous eras, and hinting that they existed even further back in history, suggests that the disorder is both timeless and universal, a neurological dysfunction that has long been present in the human population. But even if hyperactive behaviour represents a genetic mutation or variation, such variations only prove to be beneficial or detrimental under certain conditions. For example, hyperactive, impulsive and aggressive behaviour might prove inappropriate in a rigid classroom environment, but when a child is on a football pitch, or when a young adult is on the field of battle, such behaviour might be interpreted as being energetic, creative and confident. Feingold, too, saw hyperactivity as a genetic condition, but instead of focussing on the behaviour or its neurological underpinnings as the mutation, he saw the child's hypersensitivity to food additives as the crucial genetic factor. Moreover, Feingold did not necessarily view such sensitivities as a flaw; instead, he suggested that hyperactive children were overly sensitive to chemicals that might prove harmful to us all, albeit in more subtle, surreptitious ways.¹³⁷

According to sociologist Adam Rafalovich, accounts of the history of hyperactivity found in medical writing have also been shaped by a desire 'to discuss the history of ADHD as one characterizing the progress of modern clinical practice, slowly honing its nomenclature to greater levels of scientific validity and practical effectiveness'.¹³⁸ One of the problems with such present-centred and progressivist or Whiggish overemphasis on the prehistory of hyperactivity is that it overlooks the fact that most physicians and, indeed, the public were

¹³⁷ Ben F. Feingold, *Why Your Child Is Hyperactive* (New York: Random House, [1974] 1996), 13-14, 140-147, 153;

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not aware that such a disorder existed until the 1950s and 1960s. Indeed, there is no mention of hyperactive children in the first three editions of Leo Kanner's *Child Psychiatry* (1935, 1949 and 1957), the first American psychiatry textbook to focus solely on children.¹³⁹

Nevertheless, by 1966, health professionals were commenting that 'mere mention of the term "hyperkinetic syndrome" is guaranteed to stir up vigorous discussion in medical, psychological, social work, and educational circles'.¹⁴⁰ It may be true that there have always been individuals characterised by their hyperactive, impulsive and distractible natures, but it was only during the second half of the twentieth century that concern about such behaviour became widespread enough to warrant extensive medical examination, media interest and almost universal recognition of the disorder.

A further problem with Whiggish accounts of the history of hyperactivity is that they ignore the debate and discord that characterised discussion of the disorder amongst the medical community and the lay public. Much like Feingold's account of the origins of his theory of hyperactivity (discussed in Chapter 3), the 'discovery' of hyperactivity during the late 1950s is too often presented as a culmination of more than half a century of slowly accumulating scientific research into childhood behaviour. By ignoring the role of social change and professional politics, and by treating science as an unfailingly progressive enterprise, this interpretation downplays the controversy that accompanied the emergence, explanation and treatment of hyperactivity, and provides little explanation for why treatments such as the Feingold diet proved to be so popular but contentious. It also pre-supposes that hyperactive behaviour has always been thought of as pathological and undermines the social circumstances in which such behaviour is perceived to be problematic.¹⁴¹

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¹⁴¹ For example, Feingold's argument that the rise in hyperactivity diagnoses correlated a rise in food additive consumption might be true, but these two phenomena are also paralleled by many other factors, including increased educational expectations and school enrolment, more school counsellors, heightened use and promotion of psychoactive drugs and an increase in the number of psychiatric disorders generally.

Indeed, even the clinical circumstances surrounding hyperactive children described during the first part of the twentieth century were different than those of hyperactive children in the decades that followed. Most early articles which mention hyperactivity concentrated on behaviours exhibited by children suffering from readily identified conditions, such as brain injuries, infections or allergies, rather than children whose hyperactive behaviour was unexplained.¹⁴² A number of other articles, including Charles Bradley's much-cited observations on the effect of stimulants on learning, were written about children whose psychiatric problems were such that they were confined to psychiatric institutions.¹⁴³ In these cases, hyperactivity was a symptom associated with particular, pre-identified medical conditions, rather than a behaviour believed to be pathological in itself. This is one of the key distinctions between the handful of articles written about hyperactive behaviour prior to the 1950s and the thousands of articles published since.

Hyperkinetic Impulse Disorder

If it can be argued that the children described in the prehistory of hyperactivity as having 'defect of moral control', 'post-encephalitic disorder', or 'minimal brain damage' differed from more recent hyperactive children in terms of the epidemiology, aetiology and clinical circumstances underlying their conditions, when did our modern notion of hyperactivity emerge? According to the bibliographic record, as well as observers such as Feingold, the first in a great wave of medical articles about hyperactivity was published in 1957 when

¹⁴² W. Ray Shannon, 'Neuropathic Manifestations in Infants and Children as a Result of Anaphylactic Reaction to Foods Contained in Their Dietary', *American Journal of Disease of Children* 24 (1922), 89-94; Wilmot F. Schneider, 'Psychiatric Evaluation of the Hyperkinetic Child', *Journal of Pediatrics* 26 (1945), 559-70;

¹⁴³ Charles Bradley, 'The Behavior of Children Receiving Benzedrine,' *American Journal of Psychiatry* 94 (1937), 577-85; C. Keith Conners and Leon Eisenberg 'The Effects of Methylphenidate on Symptomology and Learning in Disturbed Children', *American Journal of Psychiatry* 120 (1963), 454-64, at p. 458; J. Gordon Millichap, 'Drugs in Management of Hyperkinetic and Perceptually Handicapped Children', *JAMA*, 206 (1968), 1527-30, at p. 1529; John S. Werry, 'The Use of Psychotropic Drugs in Children', *Journal of the American Academy of Child Psychiatry* 16 (1977), 446-68, at p. 452.

Maurice Laufer and Eric Denhoff, along with Gerald Solomons in one paper, published two articles on ‘hyperkinetic impulse disorder’.¹⁴⁴

On the surface, there was nothing particularly unusual about Laufer, Denhoff and Solomons’ research. They had all worked at Bradley Home under Charles Bradley and conducted their research on residents being treated there for ‘psychoses, neuroses and behaviour disorders’.¹⁴⁵ Bradley appears to have been their chief inspiration, as might be expected, since they did not cite other potential influences such as Clouston, Still, Kahn or Cohen. Like Bradley, they recommended the use of stimulants for hyperactive patients and used electroencephalogram (EEG) images to explore the brains of their patients.¹⁴⁶ There were three key differences, however, between how they described the children they were treating and how earlier researchers had done so.

First, and most importantly, Laufer and his colleagues restricted their attention to a narrower range of behaviours than their predecessors.¹⁴⁷ Although they listed ‘hyperactivity; short attention span and poor powers of concentration; irritability; impulsiveness; variability [of behaviour and school performance]; and poor school work’ as characteristic of such children, they stressed that ‘hyperactivity is the most striking item’.¹⁴⁸ As paediatrician Howard Fischer noted recently in the *Journal of Pediatrics*, there are only minor differences between Laufer and his colleagues’ conception, description and understanding of hyperkinetic impulse disorder in 1957 and what is believed about hyperactivity or ADHD today.¹⁴⁹ Moreover, they created a new name for the disorder which reflected this emphasis, namely, ‘hyperkinetic impulse disorder’. In so doing, Laufer *et al* were the first researchers to draw special

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attention to hyperactivity as a core cause of behavioural and scholastic difficulty. The label enabled subsequent researchers to focus on a specific, yet easily applicable, constellation of behaviours which they could then identify, diagnose and treat.¹⁵⁰ As child psychiatrist Justin M. Call suggested nearly two decades later, the ‘label of hyperactivity owes its popularity to the soothing effect such simple conceptions have upon issues of great cognitive complexity’.¹⁵¹

Second, Laufer *et al* departed from previous researchers in that they stressed the ubiquity of hyperkinetic impulse disorder. Despite the fact that their studies concentrated on institutionalised children, they emphasised that hyperkinetic impulse disorder was ‘very common’. Indeed, among the fifty children they sampled from Bradley Home’s population, thirty-two ‘presented the symptompicture of hyperkinetic impulse disorder’.¹⁵² Furthermore, the authors implied that the difference between children with such a diagnosis and their undiagnosed companions might be difficult to determine:

One striking point is that the characteristics which have been described are to some extent normally found in the course of development of children. That is, as compared with adults, children are hyperkinetic, have short attention span and poor powers of concentration, and are impulsive ... In the course of their development, they outgrow this mode of behavior and actually, in the course of time, so do most of the children with the hyperkinetic syndrome.¹⁵³

¹⁵⁰ The term Laufer and his colleagues derived can be construed as what historian of science Ilana Löwy has called a ‘loose concept’, one that contains elements of fluidity and indeterminacy. In Löwy’s case, the concept of ‘self’ (or biological individuality) in immunology was loose enough to have ‘facilitated interactions between scientists and physicians belonging to distinct scientific traditions’. Löwy proceeds to state that ‘imprecise concepts may help to link professional domains and to create alliances between professional groups’. This appears to be the case in the history of hyperactivity as physicians representing a number of disciplines (paediatrics, psychiatry, general practice, and in Feingold’s case, allergy) were able to interact successfully with psychologists, educators and social workers to legitimise the concept of hyperactivity and validate the means by which to treat it. Ilana Löwy, ‘The Strength of Loose Concepts - Boundary Concepts, Federative Experimental Strategies and Disciplinary Growth: The Case of Immunology’, *History of Science* 30 (1992), 371-96, at pp. 371-3.

¹⁵¹ Justin M. Call, ‘Some Problems and Challenges in the Geography of Child Psychiatry’, *Journal of the American Academy of Child Psychiatry* 15 (1976), 139-60, at p. 156.

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The hyperkinetic children Laufer *et al* described, therefore, had far more in common with ‘normal’ children than did the decidedly disturbed and violent children described by Still and those researching post-encephalitic disorder. Although they did not discuss epidemiology more generally, Laufer *et al* stressed that hyperkinetic children were usually of ‘normal intelligence’, and described how the disorder would affect children in a mainstream classroom, thus suggesting that the disorder would not be restricted to children in a psychiatric hospital.¹⁵⁴ In so doing, they also emphasised how such a disorder would contribute to educational problems and that treatment would improve academic achievement. Accordingly, hyperactivity was an educational, as well as a behavioural, disorder.

Finally, Laufer *et al* unwittingly returned to Still’s chief conundrum, which had been ignored by researchers studying post-encephalitic disorder and minimal brain damage. Specifically, if obvious neurological damage from trauma or infection was only causing some of the behavioural problems they observed, what was causing it in the other cases?¹⁵⁵ Of the thirty-two children in their sample, only eleven (thirty-four per cent) ‘had a clear-cut history of commonly accepted factors capable of causing brain damage, such as head injury, encephalitis or meningitis early in life’.¹⁵⁶ The authors postulated that neonatal difficulties and ‘purely emotional cause[s] might help to explain the aetiology of hyperkinetic impulse disorder’, but emphasised that their thoughts on the subject were merely speculative.¹⁵⁷

Unlike many of the disorders typically cited in the prehistory of hyperactivity, the disorder that Laufer *et al* described had the potential to become a widespread phenomenon. Although it represented a smaller range of symptoms, such behaviour was more common and it was not seen to be found only in brain-damaged children. Operating within a predominantly

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psychoanalytic psychiatric paradigm, the authors also integrated psychoanalytic terminology, theory and treatment (psychotherapy) into their exposition of hyperkinetic impulse disorder, thus making the disorder acceptable and treatable for both biological psychiatrists and psychoanalysts.¹⁵⁸ Unlike the previous incarnations of hyperactivity cited in medical texts, hyperkinetic impulse disorder could be applied not just to a small number of severely disturbed children, but to a large percentage of the child population.

‘Post-Sputnik Panic’: Cold War Politics and the Proliferation of Hyperactivity

The 1957 papers of Laufer *et al* provided a point of departure for modern conceptions of hyperactivity by depicting the disorder as one that could be applied to millions of children. Following their publications hundreds of researchers began exploring the phenomenon of hyperactivity. Nevertheless, sweeping categories and novel labels do not automatically attract patients or researchers. As historian Joan Jacobs Brumberg has demonstrated with anorexia nervosa, and anthropologist Allan Young has shown with PTSD, popular psychiatric disorders tend to reflect contemporary politics and circumstances.¹⁵⁹ Therefore, it is important to address why such a category became so applicable to such a large number of American children during the 1960s. Hyperactivity might have been a barely acknowledged condition in 1957, but by 1968 ‘hyperkinetic reaction of childhood’ had been added to the second edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-II)*, and medical journals regularly published articles on the disorder.¹⁶⁰ More importantly, a

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¹⁶⁰ American Psychiatric Association: Committee on Nomenclature and Statistics, *Diagnostic and Statistical Manual of Mental Disorders* 2nd ed. (Washington, D.C.: American Psychiatric Association, 1968). Social work researchers Herb Kutchins and Stuart A. Kirk have written about the expansion of the *DSM* and the psychiatric disorders found within. Although their argument emphasises the political motives of the American Psychiatric Association (APA) in promoting the ubiquity of mental illness in American society, they also emphasise how current events and social factors also influence the acceptance of disorders such as the influence of the Vietnam War on the emergence of PTSD. A good indication of the explosion of research interest is found in Winchell’s 1975 bibliography of hyperactivity research. As Winchell describes: ‘During the last decade the syndrome of hyperkinesis in children has received a tremendous amount of attention from physicians, educators, parents, legislators, and the general community. This overwhelming interest is reflected in the accelerated rate of publication in both popular and professional literature.’ Winchell, *The*

perception had emerged amongst most physicians, educators, politicians and parents that a significant percentage of American children were hyperactive and required psychiatric help.

There were many reasons why such a perception became prevalent, but one of the chief catalysts was concern about the effectiveness of the American education system and the prospect of American students during the Cold War. During the post-war period the American education system was suffering from stresses emanating from a multitude of sources. The baby boom that followed the Second World War, in particular, added approximately 75 million children to the American population during the period 1946-1964.¹⁶¹ These numbers overloaded a school system that was already suffering from infrastructure deficits incurred during the Great Depression and the Second World War, and coping with teacher shortages, as many female teachers, in accordance with general trends amongst women during the late 1940s and 1950s, stayed out of the profession or left to marry and bear children at a young age.¹⁶²

As contemporary education commentator Paul L. Gardner described, ‘in these days of crowded classrooms, expanding enrolments, and the rapidly changing world in the complex society of today, teachers across the land are hard pressed to deal adequately with their responsibilities for the welfare of their students’.¹⁶³ Moreover, researchers acknowledged that a direct link existed between overcrowding and behavioural and academic problems. For example, Laufer *et al* asserted that overcrowding could cause difficulties for children who had a tendency towards hyperactivity and distraction, as well as their beleaguered teachers:

in the crowded classrooms of today, the teacher often becomes hostile to the child who, despite seemingly good intelligence, can not sit still, can not keep

Hyperkinetic Child, vii.

¹⁶¹ Ironically, the women who gave birth to the Baby Boomers, the largest cohort in American history, were members of the smallest cohort born during the twentieth century, specifically, those born during the 1930s.

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his mind on his work, hardly ever finishes the assigned task and yet unpredictably may turn in a perfect paper. ... The child frequently fails to gain a proper foundation for the fundamentals of schooling so that each successive year he falls progressively behind.¹⁶⁴

Although the authors assumed that hyperactivity was a pre-existing condition which was exacerbated by the crowded classroom, as well as the hostility of the overworked teacher, it could also be argued that the reverse was more accurate. In other words, children taught by a stressed teacher in a teeming classroom were more likely to be troublesome, and to be singled out as such.

The impact of the baby boom generation on the school system, however, was not simply due to the numbers of children entering the school system. Historians Steven Mintz and Susan Kellogg argue that American society during this period was ‘filiarchal’, that is, dominated by and greatly concerned with American children.¹⁶⁵ The interpretation of Mintz and Kellogg was echoed by some psychiatrists of the time, including Franklin Ebaugh, who had researched post-encephalitic disorder during the 1920s. Ebaugh cautioned against the ‘child-centered’ American culture and urged that the whims of children not overshadow the needs of society. In his view, over-indulgence created ‘no more than a permanent “child,” a psychological cripple perennially seeking meanings on the prairies of Beatnikville, instead of fulfilling his future in Communityville’.¹⁶⁶ In other words, children had to be educated to serve society, rather than their own egocentric desires.

Against the background of the Cold War competition with the Soviets for ideological, intellectual, physical (for example, in the Olympics) and technological (especially military) superiority, the success of the baby boom generation was believed to be particularly crucial to

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American security and future prosperity¹⁶⁷ Such concerns were made manifest in the elevated expectations of academic achievement which accompanied the baby boomers into the classroom. Not only were more students expected to complete high school and go onto post-secondary education, an American trend which had started during the early part of the century, but there were also demands that students should achieve higher standards in order to graduate.¹⁶⁸ Students who in previous decades would have left school in their early teens for unskilled labour were now expected to attain higher levels of education. These expectations applied not only to middle-class students in the burgeoning suburbs, but also to poorer students in the slums of the American cities.¹⁶⁹ The pressure to attain high levels of schooling was partly due to parental and societal expectations, but was also due to the perception that workers would require more education to cope with technological advances in the workplace. According to research presented to the American Psychopathological Association in the late 1960s, ‘as a result of increasing emphasis on academic credentials as prerequisite to occupational success, years of schooling have been continuously prolonged’. Researchers claimed that ‘current pathways of vocational development are encumbered with hurdles that make the transition to work seem more like an obstacle course than a choice of desirable alternatives’.¹⁷⁰

The heightened expectations for American academic achievement were due to a number of factors, including the looming influence of the Cold War. The Soviet launch of two *Sputnik* satellites in 1957, for example, signalled to American politicians, educators and scientists that they might be losing the so-called ‘brain race’, and that changes to the education system were

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¹⁷⁰ Eli Ginzberg and Marcia Freedman, ‘Problems of Educational and Vocational Development in Adolescence’ in Joseph Zubin and Alfred M. Freedman (eds.), *The Psychopathology of Adolescence* (New York: Grune and Stratton, 1970), 79-85, at pp. 79-81.

warranted.¹⁷¹ The impact of *Sputnik* on the psyche of American educators and politicians was neatly illustrated in Steven A. Modée's poem, *Post-Sputnik Panic*:

when the bears
 hurled a spaceball
 into heaven
 from left field
 us got real scared
 us expanded our spaceball program
 us expanded our vocabulary too
 us expanded everything
 'till us then got the man in the moon
 hah!
 us beat them bears
 yep!
 us showed them bears
 a giant leapfrog for all mankind¹⁷²

Modée's poem captured not only the fear that *Sputnik* instilled in Americans, but also the sense of academic inferiority which accompanied the launch of the satellite. As such, many conservative educators and politicians identified the education system as the scapegoat for American intellectual shortcomings.

One of the critics' targets was the prevailing progressive education movement, envisioned by philosopher John Dewey (1859-1952) and characterised by democratic, experimental, egalitarian, and above all, child-centred learning. Historian of education Diane Ravitch has contended that by the 1940s progressive education was 'the dominant American pedagogy, ... the conventional wisdom, the lingua franca of American educators'.¹⁷³ In theory, progressive education sought to provide children with practical, tangible experiences in which they would learn skills and knowledge to prepare them to be productive members of American society.

¹⁷¹ The connection between *Sputnik* and increased diagnoses of hyperactivity was observed by some contemporary observers, for example:

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They might, for example, learn about biology, mathematics and economics by growing vegetables on school property and then selling them at a market to teachers and parents. Teachers in a progressive classroom had to be highly skilled and educated in order to ensure that such experimental projects resulted in learning and not chaos. As a March of Time newsreel of the late 1940s described, for example, teachers were the ‘keystone of Progressive Education ... Necessary qualifications: ingenuity, patience, a thousand eyes, great physical endurance!’¹⁷⁴ In practice, however, many progressive classrooms were often perceived to be disordered and aimless and, after the launch of *Sputnik*, critics of progressive education increased their calls for a return to more strict, subject-centred, authoritarian and demanding classrooms.¹⁷⁵

Critics such as Admiral Hyman Rickover (1900-1986), former Harvard president and ambassador to West Germany James Conant (1893-1978), Northeastern University president Asa S. Knowles (1909-1990) and pioneering physicist Lloyd Berkner (1905-1967), for example, all stressed that the American education system was losing pace with that of the Soviets and that more should be expected of American students.¹⁷⁶ As Rickover warned, ‘the

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¹⁷⁵ According to Barbara Ehrenreich and Deirdre English, another related target of the education critics was the permissive child-rearing philosophy espoused by paediatrician Benjamin Spock (1903-1998).

¹⁷⁶ Both contemporary critics of progressive education and historians who have sympathised with them have claimed that, by the time of *Sputnik*, progressive education was already out of step with American society and suffering from ‘old age’. Ravitch, for instance, has argued that progressive education had already ‘deteriorated into a cult whose principles were taught as dogma and whose critics were treated as dangerous heretics’ and that ‘neither the Russians nor the critics killed progressive education’. Other historians, such as Joel Spring, however, have contended that progressive education was not irrelevant, but a victim of a shift in American education policy from seeing education as a means to prepare students to protect their political, social and economic rights, or, to safeguard democracy and strive for social justice, to seeing education as means to provide for the needs of corporations and American foreign policy interests. In this way, Spring’s analysis of the shift in American education policy during this period foreshadows some of Harry Hendrick’s arguments about how children were viewed as an investment for the future by the New Labour government of Tony Blair. Asa S. Knowles, ‘For the Space Age: Education as an Instrument of National Policy’, *Phi Delta Kappa* 39 (1958), 305-10; James Bryant Conant, *The American High School Today: A First Report to Interested Citizens* (New York: McGraw-Hill, 1959); Rickover, *American Education*, 57; Joel Spring, *The Sorting Machine: National Educational Policy Since 1945* (New York: Longman, 1976), 1-4; Ravitch, *Troubled Crusade*, 79; J Gerald L. Gutek, *Education in the United States: An Historical Perspective* (Englewood Cliffs, New Jersey: Prentice Hall, 1986); Joel Spring, *The American School, 1642-1990: Varieties of Historical Interpretation of the Foundations and Development of American Education*, 2nd ed. (White Plains, New York: Longman, 1990), 322-3;

schools are letting us down at a time when the nation is in great peril. To be undereducated in this trigger-happy world is to invite catastrophe.¹⁷⁷ Knowles concurred, stating that: ‘This sphere [Sputnik] tells not of the desirability but of the URGENT NECESSITY of the highest quality and expanded dimensions of the educational effort ... the future of the twentieth century lies in the hands of those who have placed education and its Siamese twin, research, in the position of first priority.’¹⁷⁸ Knowles’ comments also reflected the contemporary perception that young Americans were unprepared for the demands of the increasingly automated workplace, which would require highly educated workers.¹⁷⁹

The alarm of critics such as Knowles and Rickover did not go unheeded. The National Defense Education Act (NDEA) of 1958, seen by most observers as a direct reaction to *Sputnik*, invested one billion dollars to improve the teaching of science, mathematics, English and foreign languages at all levels of schooling, to hire guidance counsellors and to encourage student achievement.¹⁸⁰ The combination of higher standards for academic achievement and a rejection of child-centred progressive education meant that classrooms became more demanding, especially for underachieving students. As Dorothy Barclay, parent and child editor for the *New York Times* described:

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¹⁷⁸ Capitals in original.

¹⁷⁹ Another, more subtle, factor which might have increased academic expectations for the baby boom generation was the GI Bill of Rights of 1944. The GI Bill, which originally applied to Second World War servicemen, and later to veterans of the Korean War, provided funding for education and training, and by 1956, 7.8 million servicemen had participated. Although sociologists Evan Schofer and John W. Meyer have called the addition of these students a ‘blip’ in the overall twentieth-century trend towards higher education, the re-education of such veterans, many of whom were likely the first in their family to achieve a post-secondary education, instilled an expectation that their own children, the baby boomers, would also attain post-secondary education. Ralph W. Tyler, ‘New Trends in Education’, *American Journal of Psychiatry* 122 (1965/1966), 1394-8, at pp. 1394-5; Stafford L. Warren, ‘Implementation of the President’s Program on Mental Retardation’, *American Journal of Psychiatry* 121 (1964/1965), 549-54; Bernstein, *Promises Kept*, Evan Schofer and John W. Meyer, ‘The Worldwide Expansion of Higher Education in the Twentieth Century’, *American Sociological Review* 70 (2005), 898-920, at p. 899;

¹⁸⁰ As the NDEA reached its fiftieth anniversary, the global political situation spurred calls for a new such act. The Association of American Universities, for example, has warned that ‘as the scientific and technological advantage that the U.S. has held over other nations is slipping away ... [to] rapidly developing economies, particularly in Asia’ a new NDEA is required ‘to enhance the pipeline of U.S. students trained in fields vital to our national and economic security’. The fields mentioned mimic those identified in 1958, namely, ‘science, mathematics, engineering and languages’.

The school picture ... in 1958, reflected almost entirely a tightening-up. But in some classrooms or communities, unfortunately, it was more like a cracking down. Concern about college admissions and general anxiety about America's technical ability, as highlighted by the space race, combined to produce demands for higher standards of achievement in the upper elementary grades and in high schools. The switch has given new incentive to some youngsters, but, where misapplied, its sudden severity has put a strain on others who have been unable, through lack of adequate preparation, to meet the new demands. ... Even more significant to the average family, however, is the amount of attention being given to smoking out and stimulating the efforts of the under-achievers. These youngsters of varying abilities who are not working up to their potential.¹⁸¹

The tone of Barclay's description of the post-*Sputnik* educational scene indicated that she was not completely comfortable with the 'cracking down' on schools and students demanded by critics of progressive education. Many American educators and observers agreed that 'the Soviet firing of Sputnik into space seemed to unloose a veritable Pandora's box of criticisms of us', and that other issues, for example school segregation and other civil rights issues, were more important.¹⁸² Others, such as political scientist Lewis A. Dexter, believed that forcing underachieving children to stay in school contributed to inefficiency in the school system, and that school counsellors should counsel children to leave school early for employment if they were not benefiting from education.¹⁸³ Such misgivings would foreshadow subsequent debates about the validity and epidemiology of hyperactivity.

Writing in 1959, two years prior to when Ritalin was first marketed to children, however, Barclay was not aware of the irony inherent in her phrase 'smoking out and stimulating the efforts of the underachievers'; indeed the behaviours most often associated with underachieving youngsters during the post-*Sputnik* period were those connected with

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¹⁸² Many of these issues were reflected in the New Frontier and Great Society legislation of the Kennedy and Johnson presidencies, respectively. Alice V. Keliher, 'You, the Psychologist and the Child', *Grade Teacher* 74 (1956-1957), 143; Bentley Glass, 'Science and Freedom', *Science* 126 (1957), 1317;

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hyperactivity which, in turn, would increasingly be treated with stimulant drugs.¹⁸⁴ Concern about impulsivity and hyperactivity echoed a shift regarding which behavioural characteristics were deemed to be most pernicious by American educators, physicians and politicians. Whereas shy, withdrawn and neurotic children who tended to be inactive were of greatest concern prior to the late 1950s, the increased premium on intellectual achievement following the launch of *Sputnik* meant that the most acute apprehension swung to excessively active children.¹⁸⁵ As child psychiatrist Gregory Rochlin noted, commenting on the previous trend, ‘motor activity in the young child, even if excessive, is more favourably regarded than its opposite. Although the child who is hyperactive may be as emotionally disturbed as the shy inhibited child, the latter is apt to receive more attention than the former.’¹⁸⁶ Another indication of this shift in perception is evident in Katherine Reeves’ ‘The Children We Teach’ series in the education periodical *Grade Teacher* which changed its focus from shy, withdrawn children to concentrate on children like ‘Charles’ who ‘slips from one interest to another, intense in his preoccupation of the moment, absorbing the essence of each, but moving insatiably from one activity to the next’.¹⁸⁷

¹⁸⁴ As mentioned above, Bradley had discovered in 1937 that stimulants could help raise attention levels in children. Ritalin, the most commonly prescribed stimulant drug for hyperactivity, was only made available for use in children in 1961. It was patented by Ciba seven years earlier and, ironically, was marketed as a ‘pep pill’ for much older patients, particularly ‘troublesome, miserable old people’. The drug was also recommended for residential psychiatric patients, particularly ‘markedly deteriorated chronic schizophrenic patients’. The fact that the drug was not used to treat hyperactive children initially, despite Bradley’s discovery, is further evidence that hyperactivity was not seen as a major psychiatric concern until the late 1950s and early 1960s. Anonymous, ‘New Drug Rouses Mental Patients’, *The Science News-Letter*, 68 (1955), 184; Anonymous, ‘Drugs Check Oldsters Behavior Problems’, *The Science News-Letter*, 68 (1955), 373; Anonymous, ‘Drugs Help Oldsters’, *The Science News-Letter*, 69 (1956), 68; Chauncy D. Leake, ‘Newer Stimulant Drugs’, *American Journal of Nursing*, 58 (1958), 966-8; Nicolas Rasmussen, *On Speed: The Many Lives of Amphetamine* (New York: New York University Press, 2008), 136, 156.

¹⁸⁵ Sarah Hayes has discussed how both ‘rabbits’ (neurotic children) and ‘rebels’ (delinquent children with tendencies towards criminal or immoral behaviour) were of equal concern to psychiatrists in Britain during the interwar period. Sarah Hayes, ‘Rabbits and Rebels: The Medicalisation of Maladjusted Children in Mid-Twentieth Century Britain’, in Mark Jackson (ed.), *Health and the Modern Home* (New York: Routledge, 2007), 128-52.

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Another example of how impulsive and hyperactive behaviour was becoming linked to American underachievement was illustrated in a study published in the periodical *Exceptional Children* intended to address the ‘great concern about the use of talent in our society’ and the ‘wastage in the [education] system’.¹⁸⁸ The authors compared impulsivity rates in ‘underachievers’ and ‘future scientists’ (students who had been accepted into a summer space camp) and discovered that the ‘future scientists’ were not only much less impulsive than their underachieving classmates, but also more able to control their motor activity or, in other words, less hyperactive.¹⁸⁹ The study’s conclusion was that the impulsive, hyperactive behaviour displayed by the underachieving students was the key distinction between them and the ‘future scientists’ desired by critics such as Conant and Rickover. Other researchers, such as a group led by Montreal psychiatrist Klaus Minde, recognised that hyperactive children had difficulty with the ‘increasing emphasis placed on abstract concepts, the need in the higher grades to reflect and attend rather than act impulsively on presented academic material’. They added that ‘multiple failures have tended to undermine individual children’s ambition and causes a profound sense of failure and lack of motivation – facts hardly conducive to learning’.¹⁹⁰

Increased concern about hyperactivity was also reflected in other ways. A series of Kellogg’s breakfast cereal advertisements in *Grade Teacher*, for instance, featured a trio of troublesome children, all of whom displayed different symptoms of hyperactivity. While ‘Window-Watchin’ Wendy’, who ‘skips class right in her seat’, represented the inattentive child, hyperactive children were characterised by the ‘restless and irritable’ ‘Lemon-Drop Kid’. Finally, the ‘Clockwork Kid’, who was liable to be the ‘mainspring of a classroom rebellion’,

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¹⁹⁰ K. Minde, D. Lewin, Gabrielle Weiss, H. Lavigne, Virginia Douglas and Elizabeth Sykes, ‘The Hyperactive Child in Elementary School: A 5 Year, Controlled, Followup’, *Exceptional Children* 38 (1971-1972), 215-21, at p. 221.

embodied the impulsive, defiant child.¹⁹¹ According to Kellogg's, however, these children did not need a re-vamped educational system; all they required was a better breakfast, ideally one found in a Kellogg's Corn Flakes box.¹⁹² That a company such as Kellogg's had picked up on the concern about inattentive, hyperactive and impulsive schoolchildren indicates how problematic (and profitable) such behaviours were thought to be by the late 1950s.

Hyperactivity was increasingly identified in schools by the growing numbers of guidance counsellors, the hiring of whom was demanded by education critics, such as Conant, and was made possible by NDEA funding. By singling out children as hyperactive and referring them for medical treatment, counsellors connected the educational sphere, where hyperactivity was chiefly identified and found to be problematic, to the medical sphere, where it was diagnosed and treated. Conant urged that there be one counsellor for every 250-300 students and that they should 'be on the lookout for the bright boy or girl whose high ability has been demonstrated by the results of aptitude tests ... but whose achievement, as measured by grades in courses, has been low'. This description of the under-achieving student of average or above-average intelligence would become the stereotype of the hyperactive child.¹⁹³

Although the success of the American lunar landing program had ameliorated many fears about American technological competence by 1969, when Apollo 11 and its astronauts landed on the moon, domestic strife escalated concerns about American youth during the 1960s and 1970s. Anxiety about race riots, the civil rights movement, Vietnam protests and rapid changes with regards to the music, films, clothing and drugs enjoyed by young people led to

¹⁹¹ These advertisements began in the 1956-1957 volume of *Grade Teacher*.

¹⁹² Although the medical mainstream has been hesitant to accept theories linking food additive sensitivity and fatty acid deficiency to hyperactivity have, physicians have long associated more general malnutrition with behaviour problems. N. S. Scrimshaw, 'Malnutrition, Learning and Behavior', *American Journal of Clinical Nutrition* 20 (1967), 493-502; M. S. Read, 'Malnutrition, Hunger, and Behavior. I. Malnutrition and Learning', *Journal of the American Dietetics Association* 63 (1973), 379-85;

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numerous articles, commentaries and letters in the pages of psychiatric journals about the alienation, ‘normlessness’ and turmoil experienced by American youth.¹⁹⁴ For instance, conservative California educator Max Rafferty (1917-1982), decrying the ‘decline of a once noble breed’, bemoaned that the ‘worst of our youngsters [are] growing up to become booted, side burned, duck tailed, unwashed, leather-jacketed slobs; the best of our youth [are] coming into the world ... with everything blurred, with no positive standards, with everything in doubt’.¹⁹⁵

Psychiatrists tended to interpret these issues as having psychiatric origins and mounted a number of initiatives, including the launch of the *Journal of the American Academy of Child Psychiatry* in 1962, to understand, prevent and treat the mental health problems of young people.¹⁹⁶ Often these initiatives were accompanied by the support and encouragement of the American government and general public. Preventative psychiatry, for example, was a key feature of the New Frontier and Great Society policies of presidents John F. Kennedy (1917-1963) and Lyndon B. Johnson (1908-1973) during the 1960s.¹⁹⁷ In his 1963 presidential address to the APA, C. H. Hardin Branch described Kennedy’s interest in mental health, for example, as ‘dramatic and heartwarming’, representing ‘mountains of opportunity’ for psychiatrists. Indeed, during one of Kennedy’s speeches to Congress in 1963, the president stated that mental illness and retardation were the nation’s top health priorities, and that one

¹⁹⁴ Richard E. Troy, ‘Psychiatry and the Teen-Age Rebellion’, *American Journal of Psychiatry* 124 (1968), 994-5;

¹⁹⁵ Max Rafferty quoted in Louis Jolyon West, ‘Psychiatry, “Brainwashing”, and the American Character’, *American Journal of Psychiatry*, 120 (1964), 842-50, at p. 842. Rafferty discussed his concerns about American children and the perils of progressive education in a number of other publications: Max Rafferty, *Suffer Little Children* (New York: Devin-Adair, Co., 1963); Max Rafferty, *What They Are Doing to Your Children* (New York: New American Library, 1964); Max Rafferty, *Max Rafferty on Education* (New York: Devin-Adair, Co., 1968).

¹⁹⁶ For example,

¹⁹⁷ It is commonly thought that Kennedy’s interests in psychiatry and psychiatric reform were shaped significantly by his sister Rosemary Kennedy (1918-2005), who was mentally challenged and, at the age of twenty-three, was given a prefrontal lobotomy which left her in an infant-like state and dependent on institutional care. Edward Shorter, however, has suggested that the president himself had little personally invested in the cause and that the impetus behind his government’s mental health legislation were other family members, particularly his sister Eunice.

of the chief goals of American medicine should be to ‘ascertain the causes and eradicate them’.¹⁹⁸

Following Kennedy’s assassination in 1963 by Lee Harvey Oswald, and acting on ‘a groundswell of pressure for a study of the mental health needs of children’, Congress passed the Community Mental Health Centers Construction Act which funded the Joint Commission on the Mental Health of Children, a group that researched and reported on how to prevent childhood mental illness throughout the 1960s and early 1970s.¹⁹⁹ The title of the Commission’s final report, *Crisis in Child Mental Health – Challenge for the 1970s*, reflected both the urgency of the problem and the belief that the ramifications of childhood mental illness for society would last well into the future. By 1969, the ‘Medical News’ segment of *JAMA* concurred, stating that ‘12-13% of all American children have severe enough psychological problems from ages 5 through 19 to require professional attention’.²⁰⁰

Given both the psychiatric, political and societal concern about childhood mental health during the 1960s, it is not surprising that diagnoses of hyperactivity increased. By the late 1960s and early 1970s, hyperactivity had become not only the most commonly diagnosed childhood mental disorder, but also a disorder familiar to both physicians and the public. While physicians could read about the disorder in *DSM-II*, parents could learn about the ‘millions’ of hyperactive children in *Life* magazine, which devoted seven pages to the

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¹⁹⁹ The widespread concern about child mental health was also brought to psychiatrists’ attention in the previous Joint Commission on Mental Health and Illness in 1961. The assassination of Kennedy by someone whose presumed insanity might have been prevented provided Congress with additional motivation to pass the bill, which was suitably nicknamed the Oswald Bill. Reginald S. Lourie, ‘The History of the American Academy of Child Psychiatry’, *Journal of the American Academy of Child Psychiatry* 1 (1962), 196-204; Frederick H. Allen ‘Child Psychiatry Comes of Age’, *Journal of the American Academy of Child Psychiatry* 2 (1963), 197-8; Jack R. Ewalt, ‘Presidential Address,’ *American Journal of Psychiatry* 121 (1964), 1-8, at p. 7; Reginald S. Lourie, ‘The Joint Commission on Mental Health of Children’, *American Journal of Psychiatry* 122 (1966), 1280-1;

²⁰⁰ Anonymous, ‘Millions of Children Need Psychiatric Aid’, *JAMA*, 209 (1969), 356.

disorder in October 1972.²⁰¹ As awareness of the disorder increased, however, so did controversy about its incidence and validity. Moreover, the differences in opinion regarding the epidemiology of hyperactivity were stark.

In many ways such polarity was not surprising given the context of the times. Not only were there many competing approaches to psychiatry during the 1960s, specifically psychoanalysis, biological psychiatry and social psychiatry, but there were also many anti-psychiatry critics who questioned the validity of psychiatric disorder altogether. For example, French philosopher Michel Foucault attacked psychiatry as being an agent of social control, and libertarian psychiatrist Thomas Szasz railed against the notion of mental illness in the pages of *AJP* on a regular basis.²⁰² The lack of clarity regarding the meaning, validity and treatment of mental illness generally meant that emerging disorders such as hyperactivity could be interpreted in different ways. What intensified the debate was the fact that hyperactivity was a childhood disorder. While the pathologisation of childhood behaviours was seen by some as being a highly dubious development, others saw childhood as a period where the disorders of adulthood could either be sown or prevented and, therefore, saw hyperactivity as a call to action.²⁰³

In the latter category were commentators such as Camilla Anderson, who had served in California as chief psychiatrist for the world's largest women's prison, and was the author of *Society Pays the High Cost of Minimal Brain Damage in America*, published in 1972.

Anderson believed that minimal brain damage was a major factor in most cases of crime,

²⁰¹ Physicians also learned about hyperactivity from popular publications. Feingold mentioned how this *Life* article raised his interest in the disorder when he read it in 1972.

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²⁰³ American psychoanalyst James F. Masterson, Jr. (b. 1926), for example, warned that the developmental theories of Erik Erikson, which suggested that children grew out of their psychiatric problems, were incorrect and that clinicians should intervene with troubled children in order to prevent adult psychiatric problems. Psychiatrists should assume that their patient 'will not grow out of it'. James F. Masterson, Jr., 'The Symptomatic Adolescent Five Years Later: He Didn't Grow out of it', *American Journal of Psychiatry* 123 (1967), 1338-45, at pp. 1338, 1344-5.

drug abuse and welfare dependency, and was so pernicious that it warranted eugenic solutions, including the ‘need for selective population control’, ‘changing age-old laws and values regarding abortion’ and forced ‘family limitation’ through “‘the pill,’ intrauterine devices (IUD), sterilization, or whatever techniques were reliable and nonmorbid’²⁰⁴

Although Anderson’s views harkened back eighty years to the eugenics policies of the Progressive Era, she nevertheless had support from the public and some members of the medical community. Her views might have been extreme, but they nevertheless reflected a growing opinion that hyperactivity in childhood, unless treated, was an indicator of future academic and social failure.

At the other end of the continuum were the views of journalists Peter Schrag and Diane Divoky whose book, *The Myth of the Hyperactive Child: And Other Means of Child Control*, used neo-Foucauldian social control theory to claim that the treatment of hyperactive behaviour was ‘punishment ... in the guise of therapy’.²⁰⁵ The journalists believed that an ‘entire generation is slowly being conditioned to distrust its own instincts, to regard its deviation from the narrowing standards of approved norms as sickness and to rely on the institutions of the state and on technology to define and engineer its “health”’.²⁰⁶ For Schrag and Divoky, hyperactivity was as much a political issue as it was a psychiatric condition. As the authors described with regards to the disorder, the ‘new ideology and the associated techniques – screens, drugs, behaviour modification, special programmes – all serve the purpose of legitimizing and enlarging the power of institutions over individuals’.²⁰⁷ Although

²⁰⁴ Anderson’s views were seen as extreme, even by psychiatrists who stressed the importance of hyperactivity themselves, and were not particularly influential. Her somewhat anachronistic use of the term ‘minimal brain damage’ must have also annoyed, or at least confused, some readers, given her emphasis on the genetic origins of hyperactivity. It is somewhat strange, therefore, that its review in *AJP*, by prominent hyperactivity researcher, Paul H. Wender, stated that psychiatrists would find it ‘distasteful’, not because of its eugenic overtones, but because it rejected the notion that social factors could cause the disorder.

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hyperactivity was essentially political for Anderson as well, her endorsement of eugenics as a policy to rid the nation of the disorder represented the opposite end of a political spectrum.

Although the positions expressed by Schrag and Divoky and Anderson were extreme, opinions varied widely amongst physicians and the general public on the prevalence of hyperactivity during the 1970s. Most reviews of Schrag and Divoky's book, for example, were quite positive, and their work was cited by at least one psychiatrist as being 'an excellent review' of the disorder.²⁰⁸ Conspicuous by their absence, however, were reviews of the journalists' book in medical journals. Nevertheless, Schrag and Divoky's concerns were taken up soon after by non-medical academics, such as sociologist Peter Conrad and legal scholar Robert W. Jones, and a minority of physicians also questioned the high reported rates of incidence.²⁰⁹ Such views also reflected those of certain educators, such as education professor Barbara K. Keogh who questioned the notion that the disorder was caused by neurological dysfunction and was concerned that 'hyperactivity is a general and emotional word; it is a catchall for many descriptive terms, a construct lacking in precision or in specificity of defining parameters'.²¹⁰

In contrast, most physicians who researched and wrote about hyperactivity, as well as some parents whose children had been diagnosed with the disorder, warned that, despite the perception that there had been an explosion in diagnosis since the 1960s, most children afflicted remained undiagnosed and without help.²¹¹ Much depended, however, on the criteria used to define what was hyperactive behaviour. For example, psychiatrist Paul H. Wender,

²⁰⁸ One review by Judith P. Swazey, a medical historian, called Schrag and Divoky's work an example of muckraking, but emphasised that the term was not being used pejoratively. While Swazey thought that the authors were being too polemical, she nonetheless likened the controversy over hyperactivity to that that surrounded psychosurgery a few decades previous. Henry Mayer, 'The Myth of the Hyperactive Child', *New York Times*, 9 November 1975, BR1;

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²¹⁰ Barbara K. Keogh, 'Hyperactivity and Learning Disorders: Review and Speculation', *Exceptional Children* 38 (1971-1972), 101-9, at pp. 101, 104.

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often called the ‘Dean of ADHD’, suggested that if criteria for hyperactivity were set fairly loosely, incidence could easily be as high as twenty per cent.²¹² Such a high rate of incidence, however, was somewhat understandable given the fact that Wender believed that up to two-thirds of school referrals to child guidance clinics were for hyperactive children. He described his assumptions regarding children referred to him at his clinic as follows:

With no further knowledge, any preadolescent child admitted to a child guidance clinic is most probably in the category unless proven otherwise. If, in addition, one knows that a child is not bizarre or retarded and has not been recently disturbed by a presumably noxious environment, one can make the diagnosis with some certainty. This diagnostic technique lacks subtle nicety but is quite effective ... It is comparable to a technique of adult psychiatric diagnosis attributed to William Alanson White: when a patient is admitted to a hospital, determine his age. If he is less than 40, he is probably schizophrenic; if he is between 40 and 60 he is probably manic-depressive; if he is over 60 he is probably senile.²¹³

Although Wender went on to describe more precise ways of diagnosing hyperactivity, he also stated that ‘at the expense of sounding tedious I want to reemphasize that in the practical management – the diagnosis and treatment – of children with suspected MBD, the traditional diagnostic measures are of little help ... they are expensive and generally useless for practice’.²¹⁴ For Wender the history of the child was the most important diagnostic criteria, although he also admitted that the reliability of parent and teacher reports was often suspect. Ultimately, the decision rested with the physician to decide whether a child was hyperactive or not, and given Wender’s presumptions regarding the incidence of the disorder, as well as his belief that stimulant drugs were ‘overwhelmingly’ the preferred treatment, it is understandable that alarms about over-diagnosis of hyperactivity were issued.²¹⁵ It was also not surprising that some contemporary educators admitted (albeit half-jokingly) that ‘if a

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²¹⁴ The ‘traditional diagnostic measures’ to which Wender referred included the psychiatric interview, psychological testing and neurological evaluation.

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child got through our screens without something being picked up, we'd call him Jesus Christ'.²¹⁶

Conclusion

During the late 1950s, hyperactivity transformed from being an uncommonly recognised symptom associated with severely disturbed children who exhibited many other distressing behaviours to being the most commonly diagnosed childhood disorder in the United States, accounting for the majority of child psychiatry clinical referrals and thought to affect as many as one in five children.²¹⁷ The reasons for such a transformation in perception involved both scientific and social change. As aetiological explanations for hyperactive behaviour widened, encompassing not only post-encephalitic and brain-damaged children, but also children where no specific cause could be found, the number of children for whom hyperactivity might be a diagnosis expanded rapidly to include, in theory, nearly every child in the United States. Correspondingly, the labels used to describe hyperactivity also became broader and easier to apply to larger numbers of children.

Concurrently, significant demographic, political and social changes had created an environment in which it became feasible and desirable to use these labels to explain the perceived shortcomings of American children, who were thought to be compromising the geopolitical and economic success of the United States. This marriage of category and circumstance allowed the explosion of hyperactivity diagnoses to take place. Understanding the factors underlying this union provides greater insight into why hyperactivity emerged, why it became so prevalent and why clinicians such as Feingold were dissatisfied with how it was explained and treated.

²¹⁶ Fred F. Glancy, Jr., director of a learning disabilities project in Muncie, Indiana, quoted in

²¹⁷ For more on the importance of examining the emergence of medical conditions, see:

The resilience of hyperactivity as a diagnostic category in the years since might, according to Feingold, have had something to do with the presence of additives in the food supply, as well as ongoing concern about underachieving American children, but it also involved the development of an educational, medical and technological paradigm in which the academic difficulties of schoolchildren were interpreted in medical terms and believed to require pharmaceutical interventions. One of the reasons that earlier accounts of hyperactivity are at odds with the more recent history of the disorder is that, during the first half of the century, such a paradigm was not in place. Even if physicians such as Still were observing behaviours identical to those witnessed sixty years later, the political, technological, demographic and economic framework was not conducive to transforming such observations into a ubiquitous medical disorder.

Although scientific and medical paradigms can exert powerful influences on many aspects of society, including how people define, explain and deal with abnormal behaviours, they can also be fragile, vulnerable and controversial.²¹⁸ Despite the popularity of hyperactivity as a diagnostic category, the debates, unanswered questions and confusion surrounding the disorder led to competing explanations and solutions. As the following chapter explains, one of these alternatives was the Feingold diet. Feingold's hypothesis might have questioned conventional explanations and treatments of hyperactivity, but the manner in which he described his idea was not so different than textbook accounts of the history of hyperactivity that stress its universality in order to demonstrate its genetic and neurological basis. Feingold also formulated the origins of his thesis in a calculating manner so that it would appear plausible to the majority of allergists and other physicians. As with those who emphasised

²¹⁸ Ludwik Fleck, *Genesis and Development of a Scientific Fact* (Chicago: University of Chicago Press, [1935] 1979); Thomas Kuhn, *The Structure of Scientific Revolutions*, 3rd ed. (Chicago: University of Chicago Press, [1962] 1996).

the prehistory of hyperactivity and ignored its social underpinnings, Feingold re-wrote the history of food allergy in an attempt to associate his theory with those of certain scientists and not others.

Chapter 3

The Origins of the Feingold Diet According to Dr. Ben F. Feingold

On the surface, the origins of the Feingold diet are easy to trace. In *Why Your Child is Hyperactive*, Feingold dedicated the first three chapters and many subsequent passages to outlining the laboratory and clinical origins of his theory about hyperactivity. In doing so, it appears that he attempted to achieve three aims: to depict a plausible narrative for his discovery; to show that his theory was the result of years of progressive research; and, finally, to link his research with that conducted by prominent immunologists and allergists. The achievement of these aims would presumably help to convince both parents and physicians that the Feingold diet was the product of legitimate scientific investigation and was in line with the research conducted by other respected scholars and clinicians.

By devoting the introductory chapters of his book to the origins of his diet, Feingold essentially wrote the first history of his diet. The history Feingold presented in *Why Your Child is Hyperactive* concentrated chiefly on the period between 1965 and 1972, during which time Feingold first associated food additives and behavioural problems, became aware of the hyperactivity epidemic, and ultimately began prescribing his diet. Feingold also delved as far back as his experiences as a paediatric resident in Vienna during the 1920s in order to trace the origins of his theory. Feingold stressed how clinical encounters heavily informed his ideas about food additives and behavioural problems, and described many of the experiences that informed his emerging theory, but also demonstrated that he was influenced by the contemporary research of prominent scientists, as well as his own flea bite allergy investigations during the 1950s and early 1960s. Although he did not make the analogy himself, Feingold's depiction of his diet's history resembled the building of a complex jigsaw

puzzle in which the picture emerged gradually as each piece was set into place. The final piece, Feingold's realisation that food additives not only caused general behavioural problems, but were also largely responsible for hyperactivity, surfaced only when he was forced to step away from medical practice due to illness during the late 1960s and had time to reflect on medical issues outside the traditional field of allergy. Interestingly, Feingold's own interpretation of the history of hyperactivity, specifically his contention that the disorder was a post-war phenomenon and not a condition dating back into the nineteenth-century, as many other physicians believed, allowed him to associate the explosion of hyperactivity diagnoses with the emergence and proliferation of food additives during the same period. Feingold concluded his diet's history by stating that his emergent hypothesis spurred him to reject retirement and devote his final years to promoting the diet.

Feingold presented his diet's history as a story of perseverance, Sherlock Holmes-like induction and heroism. His account suggested that these attributes allowed him to investigate doggedly the nutrition-behaviour link, identify the immunological clues that explained his clinical observations and bravely forego retirement in his seventies for the sake of hyperactive children. Feingold, therefore, provided an image of himself as a diligent, talented and dedicated clinician and researcher, the sort of physician that parents would trust and colleagues admire. Feingold also represented his diet's origins as a comprehensive, conclusive and complete history. Although Feingold allowed for further investigations into the link between environmental pollutants and behavioural problems, notably what he believed to be a connection between pollution and the disturbing rise in violent behaviour in the United States, he believed that he had firmly established the association between food additives and hyperactivity and that additional investigation or clinical trials were unnecessary. From a historical perspective, the corollary to this would be that subsequent interpretations of the history of his diet were also unnecessary.

Feingold's approach to the history of his diet is reminiscent of the fixed, linear and positivist versions of immunological history provided by pioneers of modern immunology such as Jerne and Burnett. In particular, his dramatic description of how he came to the conclusion that food additives caused hyperactivity contained similarities with Jerne's story of how he arrived at his selection theory of immunity while walking across the Knippel Bridge in 1954, a story the accuracy of which has been questioned by historian Thomas Söderqvist.²¹⁹

Feingold also contended that recollections of experiences early in his career were instrumental in helping him to formulate his theory about hyperactivity and food additives.²²⁰

Similarly, Montreal-based physician Hans Selye (1907-1982) stressed how memories of his first impressions of a clinical observation lecture as an eighteen-year old medical student at the University of Prague inspired him to consider the body's generic response 'to the stresses and strains of everyday existence' and derive his theory of the general adaptation syndrome.²²¹ Recalling how 'Even now -thirty years later - I still remember vividly the profound impression these considerations made upon me at the time', Selye insisted that these memories were responsible for his decision to abandon 'classical endocrinology' and 'spend the rest of my life studying [general adaptation syndrome]' a decision, he 'never had any reason to regret'.²²² Furthermore, Selye believed that the analysis of scientific discovery was of importance to subsequent researchers, stating that it 'is of definite value to learn, by studies in retrospect, what makes a discovery little or great, for this will help to guide our efforts', and that for 'man it is doubly instructive to analyze explorations into the depths of man's nature, for here he is both the explorer and the explored'.²²³ Anderson, Jackson and

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²²³ Selye seemingly considered that his discovery of general adaptation syndrome was of 'great' importance, as did Sir Heneage Ogilvie (1887-1971), who, in his introduction to *Stress of Life* called Selye's work 'perhaps the greatest contribution to scientific medicine in the present century'. Moreover, Selye's confidence in the manner in which he came to his discovery is inherent in his quasi-biographical work *From Dream to Discovery* in which he undertakes 'a ruthless autopsy of [his] mind' in order to pass advice onto young

Rosenkrantz, however, have cautioned against relying on ‘the memory of the discipline’ and working within the historical boundaries imposed by physicians involved in medical discovery.²²⁴ In the case of immunology, accepting uncritically the belief of actors such as Jerne and Burnett, who contended that history of immunology was essentially over by the late 1960s, leads to histories that ‘convey a reassuring sense of intellectual inevitability’.²²⁵ Such histories are not only ‘sociologically atrophied’ and contextually attenuated, they also silence key historical players who might serve to alter the account presented by chroniclers such as Jerne, Burnett, and, indeed, Feingold.²²⁶

This chapter, therefore, seeks to accept the challenge put forth by Anderson, Jackson and Rosenkrantz and to analyse Feingold’s own history in the light of other available evidence, including Feingold’s earlier research, the theories of other food allergists and the opinions expressed by people who worked with and knew Feingold. This comparative exercise highlights important inconsistencies and gaps in Feingold’s version. In particular, the origins of the diet were more complicated and involved more and different actors than Feingold suggested. It also becomes apparent that in cultivating a particular genesis for the diet, Feingold attempted to negotiate a position between patients and physicians and fringe and mainstream medicine. The potential benefits, but also inherent difficulties, involved in striking such a balance help to explain the fortunes of the diet. While one would not expect a popular, 212-page self-help book to contain a comprehensive and complete list of influences, Feingold’s omissions of key actors, developments and debates in food allergy nonetheless revealed a concerted effort to ally himself with certain allergists and immunologists and distance himself from others. In order to re-interpret Feingold’s version of the history of his

scientists contemplating a career in medical research. ; Hans Selye, *From Dream to Discovery: On Being a Scientist* (New York: McGraw-Hill Book Company, [1964] 1975), 2.

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diet, this chapter commences by outlining his own interpretation in some detail. It then identifies the gaps in his version of events and attempts to depict the medical, professional and political context in which the diet emerged. The chapter concludes by speculating as to why Feingold wrote the history of his diet's origins in the manner that he did and assessing how this version affected the reception of his diet in both the fringes and the mainstream of medical practice.

From Fleas to Food Additives

One of Feingold's chief aims in writing *Why Your Child is Hyperactive* appears to have been to account for the origins of his hypothesis in a compelling, yet plausible, manner that would appeal not only to parents, to whom the book, given its title, was primarily directed, but also to physicians, whose support would ultimately be needed if the diet was to become accepted medical practice.²²⁷ As such, he narrated the story in a friendly, familiar style that would engage parents, but also included technical details and listed leading immunologists whose mention was presumably intended to impress a medical audience. The book began with the unusual account of an Oakland woman who in the summer of 1965 entered the Kaiser Permanente Medical Center where Feingold was chief of allergy, seeking treatment for an acute case of hives. Feingold overviewed the patient's medical history, examined her and conducted allergy tests which yielded negative results or, in other words, no obvious allergies. Stating that 'food additives had been a causative factor in previous cases of hives

²²⁷ Feingold's efforts in appealing to a medical audience did not, however, did not go so far as to include consistent scholarly references. When he mentioned the work of a contemporary, he typically provided his/her name only, or cited his/her name along with the journal in which their research was published or the year in which it was published. Although one might think that this was in accordance with Random House policy for popular self-help books, it should also be stated that the scholarly articles Feingold published about hyperactivity also contain few references. Incidentally, the last article he wrote, which was published in the inaugural volume of *Ecology of Disease*, was published with no references at all, but this was because Feingold died before filling in the references and the editors could not find anyone to complete the task.

that I had seen', he placed the woman on an elimination diet and, within seventy-two hours, her hives had improved.²²⁸ Ten days later, Feingold received a call from the Center's chief of psychiatry who stated that not only had the woman's hives vanished, but her aggressive and hostile behaviour, for which she had undertaken two years of psychotherapy, had also disappeared. After confirming these changes during a conference with the patient, Feingold alerted his staff to make note of similar cases in which an elimination diet altered patient behaviour, but also cautioned that what he had witnessed may have only been a coincidence.

Having captured the reader's attention with this perplexing case study, Feingold then established how this episode provided the link between his hyperactivity hypothesis and nearly fifteen years of flea bite allergy research that he had conducted prior to 1965.

Feingold stressed the logical, incremental and routine manner in which his research and clinical activities progressed, stating that 'not only in medicine, but in many fields of science, one important observation can lead to another, although they do not, on the surface, appear to be related'.²²⁹ In order to connect the lessons gleaned from flea bite allergies to his theories about food additives, Feingold described his flea research in detail. He started by stating how, in 1951, he left his private allergy practice in Los Angeles to join the Kaiser Permanente Medical Care Program in northern California as chief of its Department of Allergy, believing that the Permanente program, a system of private medical insurance, was 'a new trend in medicine' and hoping to engage in research, 'a lifelong personal ambition'.²³⁰ As Feingold set up allergy clinics at several area hospitals and established a laboratory for the preparation of allergens, he noticed that allergies to flea bites were a common complaint in the San

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Francisco Bay area. Lacking flea allergen, he inquired as to how he might procure a ‘million of the pests’ to prepare extracts and was directed to apply to the National Institutes of Health (NIH) for a grant, which he received in due course. With the help of entomologists, Eleazar Benjamini and Dov Michaeli, the newly founded Laboratory of Medical Entomology directed by Feingold and mandated to investigate how certain insects cause disease was soon ‘the proud father of a million fleas per week’.²³¹

Next Feingold identified the key finding from his flea research with respect to hyperactivity, namely, that ‘the reaction to the fleabite was induced by a low (molecular weight) chemical present in the saliva of the insect’, otherwise known as a hapten, which must combine with proteins of larger molecular weight in order to induce an allergic response. Feingold also mentioned that Noble Laureate Karl Landsteiner (1868-1943) and Merrill Chase (1905-2004) of the Rockefeller Institute, two prominent American immunologists, had demonstrated this phenomenon a few years before, in a 1945 publication.²³² He then described becoming interested in the haptenic mechanism in immune responses, specifically because ‘*the chemicals man uses as drugs and chemicals used as food additives are both low-molecular compounds subject to the same behavior as the hapten demonstrated in flea saliva*’.²³³

Feingold’s shift from researching flea bite allergies to studying the impact of food additives might be seen as a rather pronounced transition, if only because he was shifting from studying symptoms caused by insect bites to those caused by ingested chemicals.

Nevertheless, Feingold proceeded quickly to discuss how the focus of his research shifted to

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patients who were suffering from adverse reactions to food additives and drugs, such as the dye Yellow # 5 (tartrazine) and aspirin (acetylsalicylic acid).²³⁴ Aspirin sensitivity was originally the centre of Feingold's new research programme, but after reading a report by leading American dermatologist W. B. Shelley in *JAMA* that emphasised how many foods contained a salicylate radical similar in structure to that found in aspirin, he designed a diet programme that 'placed in dietary prison' such foods as well as additives that contained the salicylate radical.²³⁵ At this point Feingold described being influenced by the research conducted by a number of important allergists, including Max Samter (1908-1999), a former president of the American Academy of Allergy (AAA), Frederic Speer, who in 1958 coined the term 'allergic tension-fatigue syndrome' and prolific allergy researcher Guy Settipane, as well as London pharmacologist and future Nobel laureate in Physiology and Medicine (1982), Sir John Vane and his associate Sergio Ferreira, which all suggested that other artificial dyes and additives could also induce reactions. Feingold 'redesigned the diet once again to include *all foods* and *all drugs* that were artificially dyed; *all foods* and *all drugs* that were artificially flavoured, as well as those containing nature's salicylates', and went on to emphasise that his diet 'was no longer a "salicylate-free" diet. It went considerably beyond that early program.'²³⁶

This revised diet, which Feingold called the Kaiser-Permanente (K-P) diet, was the one Feingold prescribed to the Oakland woman with hives and many other patients suffering from itching, skin rashes and asthma.²³⁷ Feingold stated that although he heard reports about

²³⁴ Italics in original.

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²³⁶ Italics in original.

²³⁷ Feingold disliked that his name was used to describe the diet. One reason why he might have preferred to call it the K-P diet is that Kaiser Permanente remained broadly supportive of his research, despite its controversial nature. Feingold also joked that K-P also stood for 'kitchen police, of which a certain amount is required'. Feingold, *Why Your Child Is Hyperactive*, 37; Alice D. Friedman, *History of the Kaiser Permanente Medical Care Program* (Regional Oral History Office, The Bancroft Library, University of California, Berkeley, 1986), 68, www.bancroft.berkeley.edu/ROHO/projects/kaiser/index.html, accessed on 23 February 2009; Cecil Cutting, Written Correspondence, 1 April 2008.

improvements in the behaviour of paediatric patients on the diet, he was ‘an allergist, not a behaviorist’ and did not focus on these aspects, adding that he was ‘unaware of the critical situation in hyperkinesis [hyperactivity] and learning disability that was developing throughout the country’.²³⁸ As an allergist, however, Feingold was particularly concerned with the nature of the reactions he was observing. Initially he believed he was witnessing an allergic reaction, an example of ‘the defense processes of the body’ and not just ‘any form of intolerance or even dislike’, but soon ‘became a convert to the non-allergenic concept’ after reading about how Samter and Farr had ‘convincingly demonstrated that the adverse reactions to aspirin were nonallergic’.²³⁹

That Feingold stressed the ‘nonallergic theory’ in his conception of the adverse reaction to food additives and salicylate-laden foods suggests that he condoned a considerably more limited and conservative definition of allergy than that employed by controversial Chicago clinical ecologist Theron Randolph (1906-1995) or, indeed, the founder of allergy, Clemens von Pirquet (1874-1929), whose broad definition denoted ‘any form of altered biological reactivity’.²⁴⁰ As Jackson has observed, post-war debates about the definition of allergy reflected deeper ‘disputes about the meaning or evolutionary purpose of allergic reactions’.²⁴¹ Similarly, science writer and alternative medicine advocate Ralph W. Moss, who co-authored *An Alternative Approach to Allergies* (1980) with Randolph, observed that during the 1920s:

allergists ruled out many bizarre and puzzling reactions which formerly had been a valid subject for inquiry. From this point forward, allergists were divided into two camps, the ‘orthodox,’ who accepted the antigen-antibody definition and worked within its boundaries, and the ‘unorthodox’ who

²³⁸ Feingold’s use of the term hyperkinesis in the text of a book entitled *Why Your Child is Hyperactive*, might be a subtle example of speaking to both a lay and medical audience. While parents were more familiar with the term hyperactive, the official psychiatric term, as coined by *DSM-II*, was ‘hyperkinetic reaction of childhood’.

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continued to investigate reactions in which such immunological reactions could not necessarily be demonstrated.²⁴²

While Randolph, who fitted into the unorthodox category, argued that ‘allergy constituted an entirely appropriate protective response to dangers posed by widespread environmental and ecological damage’, traditional immunologists and allergists ‘claimed that allergy was simply a manifestation of immunity gone wrong’.²⁴³ In order to assess where Feingold stood on such matters, it is important to note that his hyperactivity theory emerged after Kimishege and Teruko Ishizaka’s 1967 discovery of the antibody IgE (immunoglobulin E), a substance which was demonstrated to play a central role in immediate allergic reactions and, at the time, appeared to suggest a novel way in which to test for allergies.²⁴⁴ Although Feingold never mentioned IgE in *Why Your Child is Hyperactive*, his careful differentiation between allergic and nonallergic (or what Feingold also called ‘pharmacological’ reactions²⁴⁵) suggests not only that he wanted to be perceived as a traditional allergist, but also that he wanted to

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²⁴³ Debates about whether to use a wide or narrow definition of allergy were not restricted to allergists. J. Angell James, a laryngologist, believed that ‘if we accept the term allergy as meaning “altered capacity to react” the use of the word should be extended to include all forms of hypersensitive reactions, whether antigen antibody reactions have been proved to occur in them or not. Clinically the two types of reaction are indistinguishable and occur often in the same patient. I hope that this wide use of the word will be generally adopted.’ On the other hand, distinguished pathologist Arnold R. Rich (1893-1968) was of the opinion that the term “allergy” ... has been so debauched by indiscriminate usage that it would be fortunate indeed, if it could be dropped completely from the vocabulary of science’. Arnold R. Rich quoted in

²⁴⁴ Jackson notes that the hope that tests for measuring IgE levels would replace skin testing for allergy was not fulfilled, partly because of technical difficulties, but also because it was difficult to establish base IgE levels and correlate rising IgE levels with particular symptoms of allergy. Jackson, *Allergy*, 125-6.

²⁴⁵ In Feingold’s *Introduction to Clinical Allergy*, he stated that the classification of different allergic reactions which he employs ‘does not differ fundamentally from that proposed by Gell and Coombs’. That is, the four types of allergy Feingold described (immediate, intermediate, delayed and cytolytic) are similar to the four categories set out by Gell and Coombs (anaphylactic, cytolytic, inflammatory and cellular). The classification system of Gell and Coombs was designed to encompass von Pirquet’s broad definition of allergy as ‘changed reactivity’, but also to differentiate specific types of reactions based on their aetiology. Perhaps to keep matters simple for the purpose of a more popular book, but also possibly because his opinions about allergy had moved away somewhat from the notion that von Pirquet originated and Gell and Coombs reinforced, Feingold retreated from such a broad notion of allergy in *Why Your Child is Hyperactive* and essentially divided reactions into allergic, those ‘concerned with the defense processes of the body’, and nonallergic, those for which ‘there is no natural body defense’. P. G. H. Gell and R. R. A. Coombs, ‘The Classification of Allergic Reactions’, in R. R. A. Coombs and P. G. H. Gell (eds.) *Clinical Aspects of Immunology* (Oxford: Blackwell, 1963), 317-37;

distinguish himself from those such as Tennessee food allergist William G. Crook (1917-2002) and Randolph who downplayed the role of IgE in certain food allergies.²⁴⁶ It also implies that the audience Feingold envisioned when writing about his hypothesis was represented by conservative allergists, rather than food allergists such as Crook and Randolph. While this would have implications later on with regards to the acceptance of his diet, Crook and Randolph would have nonetheless defined the phenomenon Feingold witnessed as an allergic one.²⁴⁷

Feingold's conservative stance on the definition of allergy is also evident in a 1962 article about psychological factors and allergy in which his team of researchers assessed whether there was a relationship between skin reactivity and personality in patients being treated for allergy. In this study, Feingold emphasised the distinction between allergy patients with 'true allergic disease', those who had pronounced skin test reactions to allergens, and 'nonreactive allergic' patients, whose skin test reactions were weak or non-existent.²⁴⁸ Correspondingly, Feingold found that:

weaker reactors [those whose skin test reactions were weak] tend to be more deviant on the personality inventory. Stronger reactors are able to claim an attitude of closer affiliation with society and more adequate and satisfying interactions with others. The less sensitive tend to be dissatisfied with things as they are, more complaining, and more active in their attempts to do something about their complaints than the strong reactors. These are differences related to the dimension of sensitivity to allergens and suggest that clear psychological

²⁴⁶; Jackson *Allergy*, 201.

²⁴⁷ Crook's 1975 article, 'Food Allergy – The Great Masquerader', although it does not mention Feingold specifically, stressed how food allergies could cause hyperactivity. Letters to medical journals that Crook wrote in support of Feingold's thesis, however, indicated that he was less interested in 'understand[ing] all the mechanisms involved' as he was in the fact that, 'based on what my patients tell me ... many, and perhaps most, hyperactive children can be helped by changing their diets'. Randolph, on the other hand, only seems to have mentioned Feingold once in print. In *An Alternative Approach to Allergies* Randolph discusses an autistic ten-year-old boy who was put on the Feingold diet by his parents in the hopes that it would quell his hyperactivity. Although Randolph mentioned that the Feingold diet helped the boy, he stressed that the 'more complete, personalized approach' characterised by his regime, which eliminated all non-organic food, as well as household chemicals, not only minimised his hyperactivity, but also his autistic symptoms. Crook, 'Food Allergy';

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differences may be found between those with allergies and those with nonreactive allergies.²⁴⁹

Two relevant implications arose out of this study. First, Feingold based his investigation into psychosomatic factors in allergy on his contention that allergy sufferers consisted of two distinct groups: the ‘nonreactive allergic’; and those with ‘true allergic disease’. Feingold’s distinction in this article foreshadowed how he would distinguish between allergic and non-allergic symptoms in *Why Your Child is Hyperactive*, in which he claimed that hyperactivity caused by food additives was a non-allergic phenomenon. By reporting that the non-reactive allergic tended to be more psychologically abnormal than the true allergic, Feingold’s was also implying that their non-reactive symptoms might be psychosomatic and, more specifically, hypochondriacal. Although Feingold might have re-considered this opinion following his food additive research – perhaps some of these patients were sensitive to food additives – this article nevertheless underscored his belief that for symptoms to be truly allergic, they had to be rooted in an immunologic response. Such an opinion reinforced Feingold’s position as an orthodox allergist who employed a conservative, restricted notion of allergy.

In one sense, the restricted definition of allergy that Feingold employed, which led him to emphasise the non-allergic nature of salicylate reactions, had the paradoxical effect of expanding the potential scope of the problem. This is because, by stressing that the reaction was non-allergic, Feingold implied that individuals who reacted to food additives were not idiosyncratic or immunologically abnormal, but simply more sensitive to substances that were harmful to all people, but only capable of affecting visible responses in some. One consequence of the non-allergic nature of food additive reactions was that ‘whether the patient is an adult or a hyperkinetic child, there is no natural body defense against the

synthetic additives’.²⁵⁰ In contemplating the potential threat to the unprotected humanbody from food additive ingestion over time, Feingold expanded on the ideas of Nobel Prize laureate and founder of ethology (animal behaviour studies) Konrad Lorenz(1903-1989) that, although aggression was a natural and helpful instinct in humans, ‘[man’s] own efforts have caused over-rapid change in the conditions of his life’, and meant that ‘the aggressive impulse often has destructive results’.²⁵¹ According to Feingold, ‘Lorenz’s concept of “overrapid change” applies to the intentional introduction of synthetics into what man eats and drinks; the chemical by-products in the air he breathes; the synthetic pollution of the soil in which his food is grown; the chemical wastes in his lakes, rivers and oceans.’ Such ‘overrapid change’ was also responsible for ‘*man’s steadily growing tendencies toward unprovoked aggression and violence*’.²⁵² Continuing to accentuate the scope of the problem, Feingold contended that the ‘time is now long overdue to look at these chemicals, not only in regard to the H-LDs [children with hyperactivity and learning disability] but in regard to the human species as a whole. It is time to coldly question whether or not some of them have the possibility of disrupting the normal neurological pathways.’²⁵³

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²⁵² Italics in original. It should be noted, however, that Feingold’s application of Lorenz’s thesis in *On Aggression* was fairly loose and not entirely appropriate. Although Lorenz believed that societal and technological change was contributing to destructive aggression, a topic he expanded upon in his 1973 book *Civilized Man’s Eight Deadly Sins*, he stressed that aggression was a natural impulse and that Western society’s problem with aggression stemmed not so much from its existence, but from the insufficient means by which to discharge such impulses. He suggested, for example, that human behaviourists were wise to investigate Freud’s concept of sublimation more thoroughly in order to find ways in which humans might find ‘relief of undischarged aggressive drives’ and channel aggression in positive ways, such as sport. Correspondingly, he believed that it would ‘highly inadvisable’ to attempt to eliminate aggression because it, ‘though dangerous, is nevertheless indispensable for the achievement of the highest human goals’, given its relationship to positive traits such as enthusiasm, creativity and loyalty. Feingold, who tended to conflate hyperactivity and aggression (not incorrectly, since aggression and impulsiveness are characteristics of hyperactivity), was more concerned about eliminating the sources of hyperactivity altogether, rather than sublimating such drives into other activities. In contrast, it is possible that Lorenz would have recommended finding positive ways to channel the hyperactive tendencies of children, rather than prescribing stimulant medication or elimination diets, the idea being that stemming hyperactivity in children might also retard the development of more positive characteristics. Konrad Lorenz, *On Aggression*, trans. by Marjorie Latzke (Fakenham: Cox and Wyman Limited, [1963] 1966), x, 238-44;

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Feingold's description of the food additive problem as a potentially universal phenomenon echoed views of allergists whose expansive definitions of allergy Feingold rejected. In particular, Feingold's portent about food additives and other types of chemical exposure was reminiscent of allergist Warren T. Vaughan's (1893-1944) alarming portrayal of the scope of allergic disease. Vaughan estimated in the 1930s that up to 60 per cent of the American population suffered from allergies, if minor allergic disease was counted.²⁵⁴ This contention led him to conclude that 'allergy "is no longer the exception; it is the rule"'.²⁵⁵ The corollary to Vaughan's claim, as expressed by allergists Bret Ratner (1893-1957) and David E. Silberman in 1952, was that 'all "individuals are potentially capable of developing allergy"'.²⁵⁶ Such views were also supported by allergists such as Arthur Coca (1875-1959), the founder of the *Journal of Immunology* and past president of Society for the Study of Asthma and Allied Conditions (SSAAC), who in 1943 considered 'Vaughan's figure as essentially correct if it is not possibly somewhat conservative'.²⁵⁷ Furthermore, Feingold's belief that chemical exposure was the root of so many societal problems linked his thinking to that of Randolph, who also emphasised the role of environmental pollutants in causing many chronic physical and mental illnesses.²⁵⁸

A key difference remained, however; while Randolph believed that the reactions he witnessed were allergic, Feingold argued that they were not. From a patient's perspective, such a difference might have been moot, since the root cause of the reaction was the same, as was the solution of avoiding exposure to such noxious agents. That Feingold felt strongly enough to emphasise the distinction repeatedly highlights how questions about the nature, definition and extent of allergy persisted into the 1970s, despite the discovery of IgE, and that these

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questions divided practitioners. It also underlines Feingold's desire to be perceived as a traditional allergist who was not only clinically astute, but also kept current with developments in immunological research. The mechanism behind the reaction to food additives might not have mattered to patients, but it certainly had political implications within the allergy community.

In the midst of his realisation of the potential problems associated with food additives, Feingold's own health deteriorated. He described being 'struck down' by serious illness in the late 1960s and having to cede his duties as Chief of Allergy to one of his recruits, Don German, taking up himself the title of Chief Emeritus of the Department. Feingold recalled that while recuperating and contemplating retiring to a life of orchids and travel, something unexpected happened: he discovered the hyperactivity epidemic.²⁵⁹ He then utilised the next few pages providing an historical and contemporary sketch of the epidemic, stating that 'from the serenity and safety of my apartment study high over the Golden Gate, I was alarmed, if not shocked, by the depth of the problem, the soaring incidence, the frightening but often necessary drug management, the despair noted by both parent and teacher'. Retirement no longer an option, Feingold 'launched into educating myself on the problems surrounding the hyperkinetic child'.²⁶⁰ Studying the pioneering research of researchers such as Conners, Denhoff, Laufer, Wender, Eisenberg, Douglas and others, Feingold was confused about why he never came across such high frequencies of hyperactivity while working as a paediatrician during the first half of his career (1924-1945).²⁶¹

Trying to make sense of the controversy surrounding the disorder, Feingold thought of the curious case of the Oakland woman, but also recalled an experience in 1928, when he was a

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²⁶¹ Feingold cited no psychoanalytically or environmentally-oriented researchers even though such research was still occurring. For example:

paediatric resident in Vienna at the Pirquet Clinic.²⁶² One of his colleagues, psychiatrist and neurologist Bernard Dattner, had run a seizure clinic and required that his patients maintain a strict food diary, believing that there was a correlation between foods ingested and the frequency and severity of seizures. Although Feingold assumed that the Viennese cuisine of the 1920s was not rife with additives, his recollection of Dattner's use of diet diaries nonetheless spurred him to wonder if something hyperactive children were eating was causing the epidemic. His ideas about haptens, food additives, elimination diets and unexplained behaviour changes coalesced into a theory about hyperactivity when he recognised that the use of food additives, like the meteoric rise in hyperactivity, was a post-war phenomenon. In his words, 'a Standard & Poor's graph projecting the dollar-value increase in artificial flavours looked much like a graph indicating the rising trend of H-LD for the same period'.²⁶³ Believing that the elimination diet he had been using for his other patients might help hyperactive children and, at least, do no harm, Feingold began prescribing it in late 1972 and was soon confident that his theory was correct.²⁶⁴

In this manner, Feingold presented how he had developed his hyperactivity hypothesis and elimination diet. Feingold's revelation about hyperactivity was told as a story of routine and even tedious medical research, notably fifteen years of flea experiments, punctuated by serendipitous events, such as the case of the Oakland hives patient and Feingold's recollection of Dattner's seizure clinic. The episode is reminiscent of Selye's description of the origin of general adaptation syndrome and the histories of immunology produced by immunologists that Anderson, Jackson and Rosenkrantz critique. The problem identified by

²⁶² Incidentally, Feingold was at the Pirquet Clinic in 1929, the year in which von Pirquet and his wife committed suicide. Feingold left in that year to take a teaching position at the Northwestern University School of Medicine in Chicago, although it is unclear if von Pirquet's suicide had anything to do with his decision to leave. He might have wanted to return to the United States regardless, especially given the fact that he was Jewish and many Jewish physicians were already leaving Central Europe for North America.

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Anderson *et al* is that many factors, players and circumstances are also omitted.²⁶⁵ Some of the details left out of *Why Your Child is Hyperactive* and Feingold's medical writings were fairly trivial. A 1986 interview with Alice Friedman, one of Feingold's residents during the mid 1960s, for example, revealed that the Oakland hives patient was actually the wife of a psychiatrist and that she relapsed into her abnormal behaviour after being prescribed a hypertension medication containing tartrazine.²⁶⁶ Other aspects, such as more information about Feingold's background and why he chose to work for the Kaiser Permanente Medical Care Program, while intriguing to the historian, might not have been of interest to the readers Random House had in mind.²⁶⁷

Nevertheless, many relevant details were also excluded from the book, details which help to situate the history of the Feingold diet within the history of allergy and the history of food allergy in particular. Analysis of these factors not only discloses that the context in which the diet emerged was more complex than Feingold allowed, but also suggests that by filtering these complexities out of his own account, Feingold endeavoured to assert the originality of his idea as well as distance himself from other distinctly controversial researchers working on food allergy and clinical ecology. Ultimately, this strategy backfired in that it alienated Feingold from the very allies who might have provided more support to his ideas, especially after he died.

Food Allergy and Behavioural Problems: A Long Association

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²⁶⁷ One observation that neither Feingold, nor any of his supporters, made involves the connection between food dyes and the development of psychoactive drugs during the 1950s. According to David Healy, the first antipsychotic drug, chlorpromazine, marketed as Thorazine in the United States and Largactil in Britain, had something directly in common with many artificial food colours: both the drugs and the dyes were derived from coal tar. In other words, the molecules that could turn foods Brilliant Blue or Sunset Yellow shared key structural characteristics to those that had pronounced behavioural effects. It is strange that this connection was never made by Feingold, since it would have supported his claims. To the best of the author's knowledge, no one else has made this connection, either. David Healy, *The Antidepressant Era* (Cambridge, MA: Harvard University Press, 1997), 18-19, 43-5.

Although Feingold was careful to mention certain leading immunology and allergy researchers as being influential, the impression given to the reader was that Feingold's theory linking food and behaviour was a groundbreaking, novel and isolated epiphany. This impression was created by Feingold's bafflement, in the first pages of his book, over the Oakland hives patient's behavioural improvement and reinforced by the fact that he had to refer to a 40-year-old memory of Bernard Dattner's Vienna seizure clinic in order to convince himself that there could be a link between nutrition and behaviour.²⁶⁸ A closer look at the history of food allergy research in the United States, however, makes it difficult to trust Feingold's apparent surprise at this link.

Ever since food allergy research began in earnest during the late 1910s, physicians such as Detroit paediatrician B. Raymond Hoobler had linked food allergy and nervous system disturbances including irritability, fretfulness, restlessness and sleeplessness.²⁶⁹ In a commonly-cited article, Minnesota paediatrician W. Ray Shannon claimed in 1922 that 'food proteins to which the patient has become sensitized' could cause 'extremely restless', 'introspective', 'nervous', 'high-strung', 'cruel' and 'out-of-sorts' behaviour, as well as poor school performance.²⁷⁰ Moreover, children were often the subject of researchers studying the link between allergy and behaviour; children, who in Shannon's case study, 'could not sit still' and were 'very hard to manage'.²⁷¹ Both Shannon and other 1920s researchers, including George Piness (1891-1970) and Hyman Miller, advised that 'exclusion of foods has been found far more advisable than attempted immunization' and, therefore, advised individualised elimination diets that foreshadowed the Feingold diet.²⁷² Even Feingold's

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supposition that sensitivity to salicylates was genetic was preceded by Piness and Miller's suspicion that food allergy 'may be transmitted to future generations'.²⁷³

Beginning in the 1920s, and continuing to the 1950s, many allergists described how allergies could cause behavioural disturbances, a phenomenon often described as cerebral allergy. Although many allergists concentrated on their clinical observations of cerebral allergy, rather than its specific mechanism, it was generally believed that allergic reactions to foods and other substances could cause cerebral oedema (swelling) or impaired vascular function in the brain which could, in turn, cause migraine headaches, epilepsy and abnormal behaviour.²⁷⁴ Support for the notion that allergy could cause psychological problems, though not unanimous, was quite common amongst mainstream allergists.²⁷⁵ For example, in T. Wood Clarke's 1950 survey of 171 American and Canadian allergists, 95 'assured me that they had noticed personality changes due to allergy which corrected themselves when the allergic element was eliminated'.²⁷⁶ Clarke, a consulting allergist at the Marcy State Hospital in Utica, New York, had been introduced to the notion in 1945 when Richard H. Hutchings, past president of the American Psychiatric Society and editor of *Psychiatric Quarterly*, referred to him a fifteen-year old boy whose 'attacks of acute excitement in which he would rage around the house smashing china and furniture' had the boy bound for institutionalisation.²⁷⁷ Hutchings knew Clarke from Marcy State Hospital, had read some of

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²⁷⁴ Albert H. Rowe and Albert Rowe, Jr., *Food Allergy: Its Manifestations and Control and the Elimination Diets, a Compendium with Important Consideration of Inhalant (Especially Pollen), Drug, and Infectant Allergy*, 2nd ed. (Springfield, Illinois: Charles C. Thomas, [1931] 1972), 339;

²⁷⁵ William Waddell Duke, *Allergy, Asthma, Hay Fever, Urticaria, and Allied Manifestations of Reaction* (London: Henry Kimpton, 1925); Rowe and Rowe, Jr., *Food Allergy*; Albert H. Rowe, *Elimination Diets and the Patient's Allergies: A Handbook of Allergy*, 2nd ed. (London: Henry Kimpton, [1941] 1944); Warren Taylor Vaughan, *Allergy: Strangest of All Maladies* (London: Hutchinson's Scientific and Technical Publications, 1942); Theron G. Randolph, 'Allergy as a Causative Factor of Fatigue, Irritability and Behavior Problems of Children', *Journal of Pediatrics* 31 (1947), 560-72;

²⁷⁶ Unfortunately, it is impossible to say whether Feingold received Clarke's survey.

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Clarke's work and referred the boy to him in a final attempt to prevent him from being placed in a state hospital for mental disorders. Clarke administered a full range of skin tests for various allergens and found that the boy reacted strongly to oat and wheat, as well as certain animals, pollens and dusts. He proceeded to treat him by eliminating oat and wheat from his diet and providing desensitising inoculations for the inhalant allergens. According to Clarke:

the results of removing the oat and wheat from his diet were dramatic in the extreme. Almost overnight the boy's entire character changed. From being unhappy and apprehensive he became, in a very few days, happy and co-operative. He has had no outbreaks of temper for five years. He is friendly and full of fun. He is now doing well in college.²⁷⁸

Curious as to whether other allergists had experienced similar cases, Clarke discussed the matter at the 1949 meeting of the American College of Allergists (ACA) where the officers of the College 'were unanimously of the opinion that it was a subject worthy of systematic study' and encouraged him to investigate it and report back the following year.²⁷⁹

Clarke's survey included quotations and case studies from a number of leading American allergists, including Arthur Coca, Louis Tuft (1898-1989), who like Coca was a past president of the SSAAC, and Philip M. Gottlieb, a past president of the ACA.²⁸⁰ The case study presented by Coca is particularly interesting in that it described a child who had many symptoms that would later be associated with hyperactive children, including an above average I.Q., poor attention to detail and trouble making friends: 'Despite an I.Q. of 140, her schoolwork was not entirely satisfactory. She made frequent mistakes in copying. She was "difficult" for her teachers, had a chip-on-shoulder attitude and imagined her classmates did not like her'. After 'tomato, cheese, pork, banana, mint and licorice' were removed from her

²⁷⁸ This is probably the same 'epileptic' boy, sensitive to oats and wheat, whom Clarke discussed in a 1948 article. It is unclear as to why he failed to mention the boy's epilepsy in this article, but the epilepsy might help explain why the boy was considered for institutionalisation.

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diet, ‘her schoolwork improved and her attitude toward teachers and classmates ... [became] normal’.²⁸¹ Also indicative that the notion linking mental illness to allergy was relatively respectable is the fact that the *Annals of Allergy* chose Hal M. Davison, who had written articles about cerebral allergy himself, to comment on Clarke’s paper, rather than a detractor such as Leslie M. Gay.²⁸² Davison enthused that ‘Dr. Clarke’s paper removes any possible doubt that these symptoms must be considered the direct result of allergic reactions in the central nervous system’ and that ‘children, without the foods in their diet and with the foods in their diet, are literally Dr. Jekyll and Mr. Hyde’.²⁸³

It is difficult to speculate as to whether Feingold had read Clarke’s survey in *Annals of Allergy* or not (it was also published in *Psychiatric Quarterly*). It is possible that he had a subscription to *Annals of Allergy* in 1950, since he published an article in the journal that year, and published subsequent work there on three other occasions.²⁸⁴ Regardless of whether he read the article or not, strong evidence exists which suggests that Feingold was aware that the link between food allergy and behaviour had already been researched by many allergists. A key piece of evidence involves Albert H. Rowe (1889-1970), one of the leading food allergists in the United States and a strong proponent of the allergy-behaviour connection. Rowe operated a ‘huge food allergy clinic in Oakland’, apparently earning the allergist \$193 000 after tax in 1963, just across the Bay Bridge from Feingold’s base in San Francisco.²⁸⁵ According to English psychiatrist and strong supporter of the link between food allergy and mental health, Richard Mackarness (1916-1996), Rowe ‘pioneered the elimination diet in the treatment of food allergy symptoms, and did more than anyone else to bring to world medical

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attention the wide scope of this dietary treatment, and the variety of chronic symptoms - including mental ones - it could alleviate'.²⁸⁶

Rowe, whose career spanned the late 1910s to the early 1960s and who was president of the American Association for the Study of Allergy (AASA) in 1929, began prescribing elimination diets for food allergies in the late 1920s and wrote dozens of articles and three textbooks on allergy, usually emphasising the role of food allergy.²⁸⁷ He believed that 'psychological and emotional deviations from normal frequently arise from cerebral allergy to foods', causing symptoms such as 'drowsiness, impaired ability to concentrate, confusion, depression, tenseness, and emotional instability'.²⁸⁸ Rowe was convinced that elimination diets could help 'irritable, fussy, restless, unhappy, stubborn, unfriendly, uncooperative, antagonistic, at times angry, inattentive, tense, crying, recessive, somnolent, disliked, and at times enuretic children', and in one case described a child whose 'teachers reported that the children were afraid of him, as he had such a desire to fight. He would injure his classmates. He was a spoil-sport and took joy in ruining one game after another. His excuse for fighting was that they were trying to push him around.' After a few months on the elimination diet the boy 'adjusted so well his teacher has nothing but praise, and his cry has changed from: "I hate them, they are always pushing me around," to a happy shout of: "They like me, Ma, I'm the leader."' ²⁸⁹

Given Rowe's prominence in the allergy community it is likely that Feingold read some of Rowe's publications about allergy and behaviour. Feingold's resident Alice Friedman, for instance, noted in a 1986 interview that Feingold's allergy clinics in the mid-1960s used

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²⁸⁷ Some of these include

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Rowe's 'extremely restrictive' elimination diets to treat, like Rowe, 'all sorts of allergies'.²⁹⁰ In a chapter entitled 'Management with the Elimination diet', which she contributed to Feingold's *Introduction to Clinical Allergy*, Friedman also mentioned Rowediets, albeit suggesting that they should only be used as a last resort.²⁹¹ Furthermore, the fact that Feingold and Rowe had met is substantiated in a 1951 edition of the *Journal of the American Medical Association (JAMA)*, which included a paper Feingold presented in San Francisco to the AMA's Joint Meeting on General Practice and Pediatrics. One of the speakers invited to provide commentary on Feingold's paper, 'Treatment of Allergic Disease of the Bronchi', was none other than Rowe, whose comments were printed in the *JAMA* article. While Feingold downplayed the role of food allergies in causing 'bronchial allergy disease', Rowe, in contrast, stressed that the role of food allergies in such conditions was paramount and that elimination diets were the best treatment.²⁹² Dov Michaeli, who had worked for Feingold during the 1960s, has also stated that Feingold did know Rowe, but that the two did not get along.²⁹³

The exchange between the two Bay Area allergists also revealed that Feingold was aware of some connection between allergy and behaviour, although his comments were more in the spirit of downplaying the psychosomatic nature of bronchial allergic disease, rather than endorsing the theory that allergies could cause behavioural problems in children. Nevertheless, Feingold's statement, that 'one so frequently observes children with a history of recurrent attacks of asthma or recurring attacks of bronchial allergy who will show complete adjustment in their behavior problems when their allergy is under control', suggested that he recognised some sort of 'contributory' effect.²⁹⁴ Feingold would later

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²⁹³ Dov Michaeli, Email Interview, 19 February 2007.

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participate in writing a series of articles on the topic of psychosomatic theory in allergy.²⁹⁵

The research, funded by Kaiser Permanente during the 1960s, concluded that although ‘most of [the 195 papers investigated] seem to indicate greater psychological disturbance among asthmatics than among nonasthmatics’, ‘too many research papers, however, contain a number of serious deficiencies’ and ‘few substantive statements can be made in the field because of the many critical weaknesses in the vast bulk of research performed’.²⁹⁶

Moreover, the researchers found that ‘there is strong evidence that the allergy population is far from homogeneous either physiologically or psychologically’.²⁹⁷ Feingold similarly downplayed the role of psychogenic factors as a primary factor in allergic disease in his textbook *Introduction to Clinical Allergy*.²⁹⁸

Feingold’s investigation into the psychosomatic aspects of allergy is relevant not only because it indicates his interest in the relationship between allergy and mental health, but also because allergists who believed that allergies could cause mental illness were, understandably, some of the most vocal opponents of psychogenic allergy, believing that psychosomatic theories replaced cause with effect. Allergists who were keen to pinpoint certain substances as being particularly allergenic, such as Rowe’s identification of food or Randolph’s concentration on chemicals, were also highly critical of such theories. For instance, one of the first allergists to write about mental illness and food allergy, W. Ray Shannon, stressed that the behavioural symptoms he witnessed were not psychosomatic, nor an emotional response to the distress of experiencing other allergic symptoms such as asthma or eczema, but instead ‘the result of irritation of the nervous system resulting from

²⁹⁵ Edith H. Freeman, Ben F. Feingold, Kurt Schlesinger and Frank J. Gorman, ‘Psychological Variables in Allergic Disorders: A Review’, *Psychosomatic Medicine* 26 (1964);

²⁹⁶ These comments could also be used to describe the research done to connect food additives and hyperactivity.

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anaphylactic reactions to food proteins to which the patient has become sensitized'.²⁹⁹

Furthermore, while the title of Mackarness' book *Not All in the Mind* reflected his contention that allergies could cause mental health problems, he also questioned 'emotionally determined' or psychosomatic theories of allergy, which suggests that his title could have a double meaning.³⁰⁰ Rowe, who believed that food allergy could cause behavioural problems, complained that in many cases of diarrhoea caused by food allergy 'psychogenic' factors were mistakenly attributed and that 'in one case entirely controlled by the elimination of allergenic foods, hostility to the patient's husband had been blamed!'³⁰¹

Some allergists, resistant to the notion that allergy could be psychosomatic, looked to other possible factors to explain the perceived rise in allergy. Evidently frustrated by the popularity of psychogenic allergy theories, Ethan Allan Brown, president of the AAA in 1957, decried that 'in present-day journals (the editors of which should know better) there are papers (by physicians who should also know better) stating that not only asthma, but all allergy as such is "psychosomatic"'. Brown opined that 'the less one knows of any aspect of medicine, the more likely one is to believe that it is all psychosomatic' and that 'much of this literature is excellent fiction'.³⁰² What is especially interesting about Brown's criticism, however, is his contention that food additives, not neuroses, were causing the rise in allergy. Specifically, Brown stated that 'in this age of chemicals and synthetics there is truly no limit as to what substances may be discovered as causes of allergy' and that:

it is not too much to expect that one or several new ubiquitous allergens may be discovered at any time. This would, of course, change overnight the present practice of allergy. Among these might be the more than 1,000 "additives" now ingested with foods and now certified for safety but not to allergenicity.³⁰³

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³⁰¹ In one of Rowe's textbooks he admits that psychogenic asthma was possible, but in another publication he emphasised strongly the role of food allergy in asthma.

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Although by the late 1960s Feingold was similarly concerned about food additives, he did agree that hyperactivity caused by food additives could be exacerbated by social conditions such as overcrowded living conditions and poverty.³⁰⁴ Feingold, like many allergists, did not reject psychosomatic factors completely but stressed that psychiatrists, psychologists, laymen and even allergists greatly overemphasised their impact.³⁰⁵ Clarke, for example, suggested that:

if allergists would pay more attention to the psyche of their child patients, if child psychiatrists would appreciate that psychosomatic medicine can travel in reverse gear, that physical allergy of the brain can cause emotional changes, and if the two would co-operate in the study of the 'problem child' from both the allergic and psychic angles, we may well hope that our state hospitals may not need such extensive facilities for the care of children, that many children may cease to be problems, and fewer adults become psychotic.³⁰⁶

Clarke's words were highly reminiscent of Feingold's concerns that hyperactivity might represent the proverbial tip of the iceberg and that '*man's steadily growing tendencies toward unprovoked aggression and violence*' might be due to 'the pollutants we ingest'.³⁰⁷

Feingold's concern about pollution reflected mounting fears during the 1960s about the impact of environmental degradation on human health. Perhaps the greatest catalyst in raising awareness about chemical pollution was the publication of Rachel Carson's *Silent Spring* in 1962.³⁰⁸ Carson described the overuse of agricultural chemicals as 'a strange blight', 'an evil spell', 'a shadow of death' and 'a grim spectre', which was wreaking havoc upon American ecosystems and wildlife.³⁰⁹ She also likened the effect of pesticides to that of radioactive fallout, an image with which Americans experiencing the height of the Cold War

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could closely identify.³¹⁰ Although Carson's work was attacked vehemently by industry trade groups and their supporters, such critics could not extinguish the growing notion that the American relationship with the environment had changed. The fact that a CBS television programme on Rachel Carson and *Silent Spring* attracted nearly 15 million viewers also indicated that Americans were becoming concerned that their relationship with the environment was becoming increasingly tenuous.³¹¹ As environmental historian John Clark has described, commenting on the impact of *Silent Spring*, 'once described by what it produced, Western culture is now defined by the waste and pollution that it generates'.³¹²

Feingold's concerns about 'the pollutants we ingest' tapped into many of the same fears about the affect of chemicals on health that Carson's work described. Nevertheless, Feingold was reluctant to associate himself with many of the players within the ecology movement that *Silent Spring* spawned. A telling example of this reluctance was Feingold's limited relationship with Theron Randolph, an allergist, a friend of Rachel Carson and the founder of the clinical ecology movement in the United States. Although some of the previously mentioned allergists would have retired or died by the time Feingold became interested in hyperactivity, Randolph was active and well-known during this period for his work on multiple chemical sensitivity and food allergies, including the impact of allergies on the behaviour of children.³¹³

In some ways, the two physicians lived parallel lives; they were born within six years of one another, spent time teaching medicine at Northwestern University in Chicago early in their careers (though at different times), underwent significant mid-career changes and mid-life divorces, courted controversy with their theories and inspired their adherents to carry on with

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their work after they died. Aspects of both allergists' theories were also influenced by the notion that industrial progress, and specifically the increased use of synthetic chemicals, was hazardous to the health of Americans in a variety of ways. As early as the 1940s, Randolph had begun to link chronic illness, including mental health problems, to various pollutants in the air, water and food supply and to prescribe elimination diets to his clients.³¹⁴ Although his 1951 book *Food Allergy*, co-written by Herbert Rinkel (1896-1966) and Michael Zeller, focussed on allergic reactions to common, natural foods such as corn, wheat, milk and eggs, by 1961 he had written a series of four articles for the *Annals of Allergy's* 'Progress in Allergy' feature on the topic of 'Human Ecology and Susceptibility to the Chemical Environment'.³¹⁵ It is likely that Feingold at the very least knew about Randolph's series of articles, since he published a paper on his flea bite allergy research in the same volume.³¹⁶

The question Randolph addressed in these articles was:

how much do we know about the long-term effects of such by-products of "progress" as the chemical pollutants in the air of our homes and cities; chemical additives and contaminants in our foods, water and biological drugs, as well as our synthetic drugs, cosmetics, and many other personal exposures to and occupational contacts with man-made chemicals?³¹⁷

Feingold was concerned with similar questions, arguing that:

in the evolution of man, a hundred-plus years of technology torrent are as insignificant as a polyp on a coral reef. But applied to living in the last half of the twentieth century, they are cataclysmic to behavior. Man has not had adequate time to adapt to the changes and new environment, physically or mentally. All of the changes, mechanical and chemical, have twisted the physical environment as well as the social environment out of all recognition.³¹⁸

³¹⁴ Kinney wrote and published a newsletter for Randolph's Human Ecology Study Group during the 1970s and 1980s and was instrumental in establishing the Theron G. Randolph archive at the Francis A. Countway Library for Medicine, Harvard University.

³¹⁵ Rinkel, Randolph, and Zeller, *Food Allergy*; Theron It is probable that the title of the third article in the series, 'Human Ecology and Susceptibility to the *Human* Environment' is a typo, since all the other articles are titles 'Human Ecology and Susceptibility to the *Chemical* Environment'. Italics added.

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³¹⁷ Randolph, 'Human Ecology', 518.

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Feingold's interest in the ecological aspects of his theory increased during the 1970s, as is evident by his final publication in the inaugural volume of *Ecology of Disease* in which he warned that 'in recent years the alterations in the biological profile have been accelerated by thousands of mutagenic agents provided by the increased concentration of pollutants in the atmosphere, water, soil and food'.³¹⁹

Despite their shared interests in food allergy, allergy and children's behaviour problems and exposure to environmental pollutants, Feingold never listed Randolph as an influence or even mentioned his work in passing.³²⁰ Feingold also made no citation of the work of allergists such as Rowe, Clarke or Brown, despite his interest in the psychological aspects of allergy, his proximity to Rowe and use of his elimination diets, and the fact that his observations of hyperactive children bore a resemblance to those made by other allergists. Not only did Feingold fail to mention whether earlier allergists such as Shannon, Piness, Duke, Coca or Vaughan had influenced his work, but he also omitted later research that specifically linked food allergy and hyperactivity. These included Wilmot Schneider's 1945 article, which contended that elimination diets could improve the behaviour of hyperactive children, and Fred Kittler and Deane Baldwin's highly relevant 1970 paper on the role of allergic factors in hyperactivity.³²¹ It is striking that Feingold did not mention Kittler and Baldwin's paper in any of his publications, since he would have been formulating his hyperactivity thesis

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³²⁰ Other contemporary observers, however, did associate Feingold and Randolph's work. A 1974 letter to the editor of the *American Journal of Psychiatry*, for instance, referred to both as providing evidence that chemicals in the food supply could lead to hyperactivity.

³²¹ Kittler and Baldwin did not mention Feingold, but that is probably because he did not make his findings public until 1972. It would have been unlikely for Kittler and Baldwin to have heard about Feingold's clinical efforts in California, since they were based in Arkansas. It is somewhat ironic, however, that physicians based in Arkansas were conducting research on alternative approaches to hyperactivity. This is because, during the late 1960s, Arkansas hosted a disproportionately high number of chapters of the Association for Children with Learning Disabilities, an advocacy group whose members strongly advocated mainstream, neurological aetiologies for hyperactivity and supported the use of stimulant medication. Wilmot F. Schneider, 'Psychiatric Evaluation', 567-8;

contemporaneously with their paper's publication in *Annals of Allergy*. Instead, Feingold presented the history of his diet as a development distinct from the larger history of food allergy and the long association of food allergy and mental health problems such as hyperactivity.

Conclusion

One question raised by the lack of references to the history of food allergy in Feingold's writing is whether he ignored this history on purpose. In other words, was Feingold ignorant of contemporary and earlier food allergy research or, instead, were his omissions the result of careful, strategic decisions? Analysis of *Why Your Child is Hyperactive*, Feingold's own researches and oral history evidence suggest that the latter is more likely the case. In *Why Your Child is Hyperactive*, Feingold cited a considerable amount of research, indeed more than one would suspect for a self-help book primarily targeted at parents. His inclusion of Chase and Landstenier's hapten studies, Vane and Ferreira's pharmacological research, Samter and Farr's investigations into the mechanism of aspirin sensitivity, Lin-Fiu's study into lead exposure and overviews of the current research conducted in not only hyperactivity, but also obscure conditions such as phenylketonuria, Turner's syndrome, Klinefelter's syndrome, Zurich hemoglobinopathy and Lesch-Nyhan syndrome hint that his knowledge of contemporary medical research was comprehensive.³²²

The references in Feingold textbook *Introduction to Clinical Allergy* also suggest that he was familiar with allergy studies.³²³ Although Feingold was primarily a clinician and not a researcher, he did produce publications on bronchial allergic disease, flea bite allergy and psychosomatic aspects of allergy in journals such as *JAMA*, *Annals of Allergy*, *Journal of*

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Allergy and Psychosomatic Medicine.³²⁴ These publications reflect Feingold's broad immunological interests as well as his willingness to review a wide range of medical literature. Finally, and most convincingly, Feingold's colleagues, such as Bernard Weiss, a pioneering environmental toxicologist, Dov Michaeli, a biochemist and immunologist who worked for Feingold during the 1960s, and Alice Friedman, Feingold's internist during the 1960s, have acknowledged that he was well aware of the food allergy literature and the theories of food allergists such as Randolph and Rowe.³²⁵

If Feingold was aware of current food allergy research, then, what explains his conscious decision to ignore this literature in his description of the origins of his diet? Why did he stress the influence on him of some immunologists and allergists and not others? The answers to these questions help to clarify why Feingold's diet was not particularly popular amongst food allergists and clinical ecologists, but also reveal insights into the controversial world of food allergy research and clinical practice before and after the Second World War. Specifically, Feingold's careful omission of the larger history of food allergy in the construction of his diet disassociated his theory from those of food allergists and clinical ecologists whose ideas, though popular, were also highly divisive. By ignoring the history of food allergy and focussing on more respectable research initiatives in *Why Your Child is Hyperactive*, Feingold attempted to appeal to a broader spectrum of physicians, not just food allergists and clinical ecologists. Although oral history evidence reveals that Feingold had personal and professional reasons for wanting to segregate his theory from those of more controversial physicians, it is also clear that Feingold simply held different beliefs about the definition of allergy than those held by most food allergists. It is likely that, having

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employed a conservative notion of allergy for over a quarter of a century, Feingold was unwilling to compromise his principles merely to accommodate his theory to those of food allergists for whom he had little respect. The following chapter will elaborate on the reasons why Feingold chose to downplay the role of food allergy research in influencing his theory and discuss the impact of this decision on the reception of his diet.

Chapter 4

Food Allergy: Witchcraft, Fad or Racket?

For much of the twentieth century, allergy was a controversial subject amongst medical practitioners and occupied a tenuous position on the medical hierarchy. Similarly to psychiatrists, most allergists believed that other physicians viewed their discipline as scientifically questionable, lacking in laboratory investigations and over-reliant on clinical observations. Allergists also thought that the disease they studied and treated was more widespread and was responsible for a wider array of symptoms than was commonly thought; such suspicions also mirrored psychiatric fears about the prevalence of mental illness, particularly following the Second World War. Despite these broad concerns allergists argued amongst each other about the extent of these problems and how best to deal with them.

Central to these debates were fundamental issues related to how allergy should be defined, treated and conceptualised and, due to a range of factors that will be discussed in this chapter, food allergy had the longest history of causing fractious disagreements. Allergists tended to be split on the subject of food allergy, with food allergists supporting the notion that allergic reactions to food were widespread and responsible for causing all manner of illness and orthodox allergists dismissing such notions altogether. These divisions also affected clinical practice, including what allergists suspected was causing their patients' symptoms and what treatments they offered.

Consequently, there was good reason to believe that Feingold's position on these various debates in *Why Your Child is Hyperactive* would affect which allergists would be sympathetic to his hyperactivity hypothesis, and which would be hostile to it. As an experienced and

respected allergist who not only led research programmes but also managed numerous allergy clinics in the San Francisco area, Feingold did not merely observe such debates, but actively participated in them. As such, Feingold was acutely aware of the context in which his hypothesis would have to survive, both in theory and in clinical practice. Feingold's reasons for setting the origins of his diet outside the history of food allergy, therefore, are intertwined with the broader history of food allergy and its place within allergy.

In order to understand Feingold's rationale, this chapter provides an overview of the history of food allergy, explaining how it was variously understood by food allergists and why it was such a divisive subject. It begins by describing how many allergists thought allergy was an under-diagnosed disease that required more attention from both clinicians and medical researchers. The chapter then demonstrates how food allergists believed that allergies to food were particularly widespread and proceeds to delineate the theories of the most prominent food allergists, including Albert Rowe, Warren Vaughan, Arthur Coca and Theron Randolph. The reasons why the theories of these allergists, as well as food allergy itself, were so controversial are then explored. The chapter concludes by contending that Feingold's decision not to associate his theory with those of food allergists was partly a conscious attempt to make his theory appear more plausible to orthodox allergists, but also reflected the fact that Feingold himself had been a conservative allergist who disliked the theories espoused by food allergists. Feingold's stubborn refusal to ally himself with food allergists who would have been sympathetic to his theory shows how the personalities, beliefs and allegiances of scientists can play a considerable role in affecting the reception of their theories.

Food Allergy: 'The commonest form of human allergy'?

In 1958 food allergist William G. Crook and his colleagues Walton Harrison and Stanley Crawford wrote in *Pediatrics* that despite the fact that ‘allergy celebrated its golden anniversary in 1956 ... [and] is as old, or older, than many other branches of medicine, it occupies a uniquely confusing and controversial position. No other field of medicine has been the subject of as much violent controversy, difference of opinion and confusion.’³²⁶ In lamenting the lack of respect and resources accorded to the discipline, the authors added that ‘too often allergy is regarded as witchcraft, a fad or a racket. In this respect, allergy has had to fight some of the same battles that psychiatry has been fighting.’³²⁷ Moreover, allergy’s position as the “‘stepchild” of medicine’ prevented it from competing successfully for funding with other diseases such as polio, cancer, heart disease, muscular dystrophy and multiple sclerosis, meaning ‘that during the past 10 years, more time and money have been spent on fundamental research in such a condition as cystic fibrosis of the pancreas than has been expended on the allergic diseases, even though the latter occur perhaps 100 times as frequently, and constitute just as challenging a scientific enigma’.³²⁸

It could be argued that these sorts of comments might have been expected from a controversial food allergist such as Crook, who would later heartily support the link between food allergy and hyperactivity and write *The Yeast Connection*, which claimed that various forms of yeast infections could cause unexplained symptoms ranging from headaches and earaches to severe inflammation and depression.³²⁹ Regardless, Crook’s opinion about the undeservedly low status of allergy within the medical hierarchy, as well as the controversy that surrounded the discipline, reflected the views of many allergists, including well-respected leaders of the American allergy community. Ben Z. Rappaport (b. 1897), president

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³²⁷ Suspicion about allergy and allergists had not faded away when allergy approached its centenary. Physician Theodore Dalrymple suggested in 2003 that ‘the whole idea of allergy remains slightly disreputable because it is so fertile a field for quacks who prey on hypochondriacs’. Theodore Dalrymple quoted in

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of the AAA in 1953, for example, contrasted the \$27 000 000 allocated for cancer research by the National Research Council during the period 1947-1951 with the \$800 000 provided for allergy research during the same period.³³⁰ Rappaport warned that allergists would have to ‘tell the story of allergy over and over for a long time to produce the necessary impact on public attention and opinion’ and reduce such ‘disparity’.³³¹ Horace S. Baldwin (1895-1983) and W. C. Spain (1878-1959), who both served terms as presidents of the AAA, as well as chairs of the Academy’s education committees, added that it was difficult to attract ‘high grade men’ to specialise in allergy and that it was ‘more than deplorable that many young internists have been permitted to finish their training without contact with allergy and allergic thought’.³³² Orval R. Withers, a president of the ACA, echoed such views, criticising the writers of medical textbooks for not making enough mention of allergic diseases, even though ‘10 to 20 per cent of all the persons who visit physicians’ offices are allergic’.³³³

The ‘sorry plight of allergy’ lamented by the discipline’s leaders during the post-war period was due to a number of factors.³³⁴ Mark Jackson has noted that political disputes between different groups of allergists, such as members of the AAA and a splinter organisation, the ACA, as well as between allergists and other specialists, hampered the discipline’s ability to gain speciality status and board certification from the AMA.³³⁵ A series of acrimonious letters published in the *Journal of Allergy* between prominent allergist Louis Tuft and dermatologist Rudolph L. Baer, and moderated to a certain extent by allergist Louis Webb Hill, about whether allergists or dermatologists should treat atopic dermatitis (eczema) highlighted the disputes allergists fought with dermatologists, in particular, over clinical territory.³³⁶ Given

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the claims of allergists such as Vaughan, Coca and Rowe that allergies of one sort or another affected between 35 and 60 per cent of Americans and were responsible for a wide range of symptoms affecting the respiratory, gastrointestinal, epidermal and nervous systems, it is understandable that allergists came into conflict with physicians representing other medical specialties.³³⁷

Other leaders within American allergy looked within their own profession to assess why they lacked the respect of the broader medical community. Many allergists, for example, were troubled by the notion that allergy, much like psychiatry, was more of an art than a science. Max Samter remarked on this idea in his presidential address to the AAA in 1960, stating that ‘the art of allergy which we practice is based on tradition - the joint experience of generations of allergists. Experience, however, is only the beginning; the art of allergy must now be persuaded to adopt and perhaps to be altered by its own unruly offspring, the science of allergy.’³³⁸ In other words, Samter expected allergists to venture beyond the routine clinical practice of performing skin tests for allergy and de-sensitising patients to various allergens, and learn from laboratory investigations as well.³³⁹ Samter’s plea echoed comments made by pioneering British allergist, John Freeman (1876-1962) who cautioned against ‘basing clinical decision merely on an accumulation of cases’ and believed that the relationship between the clinic and the laboratory ought to be ‘symbiotic’.³⁴⁰

Implicit in Samter’s discussion of the reliance on tradition and experience was the suggestion that allergists could also be criticised for being one-dimensional in their thinking about allergy and somewhat close-minded when it came to new ideas. Another president of the

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³³⁹ Ironically, Samter would be making similar comments nearly twenty years later in 1979, stating that ‘the practice of allergy is virtually synonymous with immunotherapy’. Max Samter quoted in

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AAA, William B. Sherman, was thus concerned that if allergists ‘think only in terms of allergy and look at the skin tests rather than the whole patient, we may become perhaps not faddists but not the physicians we should be’. Sherman added that the more allergists ‘stick strictly to one approach, the more likely they are going to consider us faddists. I find that a good many intelligent internists still think that injecting dust into patients is a strange way to cure disease.’³⁴¹ Likewise, Freeman also supported such a holistic approach to approaching patients, believing strongly that allergists ‘must not treat human beings as mere cases. You must observe the traditional maxim of “treat the individual man” and all his special commitments at the moment.’³⁴²

One of the ways allergists attempted to become more holistic and pluralistic during the post-war period was by incorporating psychosomatic and psychoanalytic theories into the aetiology of allergy.³⁴³ Although adding such a dimension was not necessarily deemed to be unscientific within the American context of the 1940s, 1950s and 1960s, given the contemporary prevalence of psychoanalysis in western society, psychosomatic theories of allergy, as well as treatments such as hypnosis and parentectomy (removing children from asthma or allergy-producing home) also attracted heated criticism.³⁴⁴ Moreover, when psychoanalytic psychiatry was supplanted by biological psychiatry in North America during the 1960s and 1970s, psychosomatic theories of allergy could be seen as a liability to those allergists concerned about the reputation of allergy as a legitimate medical science.

Although psychosomatic theories of allergy were controversial, they nevertheless earned considerable support; while many allergists, as well as some psychiatrists, doubted that

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³⁴³ Carla Keirns, ‘Better Than Nature: The Changing Treatment of Asthma and Hay Fever in the United States, 1910-1945’, *Studies in History and Philosophy of Biological and Biomedical Sciences* 34 (2003), 511-31, at p. 521;

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mental distress directly caused allergies, a significant number, including Feingold, agreed that emotional problems could indeed exacerbate the symptoms of allergy.³⁴⁵ In many respects, food allergy was a more consistently contentious topic and it was into this tempestuous territory that Feingold entered when he wrote *Why Your Child is Hyperactive*. The divisive nature of food allergy was hinted at as early as 1916, when Hoobler described his experiences of it in *JAMA*. Seemingly aware that his observations about food allergy might arouse hostility and breach the territorial boundaries of other medical specialities, Hoobler cautiously wrote:

I am well aware that many of the symptoms named [sneezing, coughing, wheezing, vomiting, irritability, restlessness, fretfulness, insomnia, eczema] are symptoms of other very common diseases, and it is not my desire to claim that they occur only as symptoms of protein sensitization; but it is my observation that when the group of symptoms as outlined occurs and reoccurs in an infant early in its existence, one should be on guard and should carefully watch for further developments.³⁴⁶

Hoobler's reticence was not, however, reflected by subsequent food allergists. Food allergy's notoriety was likely due, in part, to the unbridled enthusiasm its proponents expressed for its prevalence and significance. Rowe's textbooks of 1931, 1937 and 1941, as well as his numerous essays, for instance, contended not only that foods 'probably produce more allergic manifestations in various tissues of the body than any other group of allergens', but also that such allergies caused an enormous array of symptoms, ranging from epidermal, respiratory and gastrointestinal problems to acute neurological reactions such as epilepsy.³⁴⁷ According to Rowe, the failure 'of the majority of physicians and specialists, including most allergists, to recognize, study, and control such allergies is in our opinion one of the main deficiencies

³⁴⁵ ; Ben F. Feingold, Frank J. Gorman, Margaret Thaler Singer and Kurt Schlesinger, 'Psychological Studies of Allergic Women: The Relation between Skin Reactivity and Personality', *Psychosomatic Medicine* 24 (1962), 195-202, at pp.195, 201; A. W. Frankland, In-Person Interview, 22 May 2007.

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in medical practice today'.³⁴⁸ Endeavouring to correct this deficiency, Rowe found opportunities to mention the scourge of food allergy, even when it was not particularly appropriate. As a discussant for a 1929 paper on the use of vaccine therapy for infectious bronchitis and asthma, he reserved comment on the authors' study and, instead, proceeded to assert that such symptoms were not the result of infection at all, but were instead caused by food allergy. The authors, who, like Rowe, were from the San Francisco Bay area, expressed exasperation in their reply:

Answering Dr. Rowe, we wish to state that our present study deals with bacterial-sensitive asthmatics and does not mention the large group sensitive to proteins and foods. We grant the existence of food allergy, but are not discussing it in this paper; in fact we thought our procedure, which was most careful and painstaking, excluded this group.³⁴⁹

Apparently, Rowe was not chastened by this response, nor the dismissal of his claims by the other discussant, given the fact that he took a similar approach to 'discussing' a paper given by Feingold in 1951.³⁵⁰

Rowe's zeal was echoed by other food allergists. In his provocatively-titled book, *Allergy: Strangest of all Maladies*, Warren T. Vaughan wrote: 'Sensitization to foods is the commonest form of human allergy.'³⁵¹ Food allergy, therefore, contributed significantly to the allergies suffered by, as Vaughan claimed, 60 per cent of the American population. In addition, the 1935 article in which Vaughan made this estimate, based on a survey of the village of Clover, Virginia, also implied that, since many food allergy sufferers were able to identify, either consciously or unconsciously, the foods to which they were allergic, they were able to remove such foods from their diet independently of medical advice. As such, the rate of food

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allergy might be even higher than his survey suggested and certainly higher than the clinical experience of most allergists would indicate.³⁵²

Vaughan's rationale for such high rates of allergy, as well as the numerous instances of allergy sufferers 'curing themselves', helps to explain why food allergy was such a perplexing subject for allergists. Specifically, Vaughan believed that food allergy sufferers could be divided into two distinct categories: major food allergy sufferers, referred to by Vaughan as 'the frank allergic', and those who suffered from minor allergy, referred to as the 'fortunate allergic'. The frank allergic, representing seven to ten percent of the general population, were distinguished not by having severer symptoms than minor allergy sufferers, but instead by their inability to identify the offending food or other allergen. Such individuals, bewildered by their symptoms, sought medical advice and represented the majority of allergy sufferers seen by physicians. The reason the frank allergic were unable to identify the source of their allergy was that they were typically allergic to staples such as 'wheat, milk, bean, egg [or] Irish potato'.³⁵³ The ubiquitous nature of such foods made it unlikely that the frank allergic would suspect them as the cause of their symptoms, let alone pinpoint which food was the offender. Moreover, the fact that the frank allergic were unknowingly allergic to such common foods meant that they might constantly be suffering from symptoms ranging from indigestion to headaches.³⁵⁴ The fortunate allergic or minor allergy sufferers, on the other hand, were allergic to less commonly consumed foods, such as 'cucumber, watermelon, strawberry, tomato, onion, and cabbage', foods which they were able

³⁵² Vaughan indicated that he did not count the ten per cent of respondents who were unable to self-identify as allergic in an effort 'to avoid hyperenthusiasm in the subject' of food allergy. Given the fact that Vaughan's estimate of the prevalence of allergy suggested that more Americans were allergic than not, his attempt at restraint likely went unnoticed.

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to identify as offensive and subsequently avoid.³⁵⁵ These patients were also fortunate, according to Vaughan, because of the possibility that, if they avoided the food allergen for a long enough period of time, they might eventually lose their sensitisation to it. The frank allergic, continually coming into contact with the unknown food allergen, were also unfortunate in this respect.³⁵⁶

Vaughan's dichotomy of the fortunate and unfortunate allergic differed greatly from distinctions made by other allergists, including Feingold, who instead divided food allergies into those that involved an immediate reaction and those that involved a delayed reaction, often occurring an hour or more following ingestion of the offending food. Feingold insisted that it was the immediacy of the reaction which was the key factor that allowed people to identify problem foods, rather than whether or not a person was allergic to a commonly or rarely ingested food.³⁵⁷ Such discrepancies were somewhat understandable, given the fact that, as Jackson has demonstrated, allergists devised many different ways by which to classify allergy, including classification by organ system affected and classification by underlying immunologic mechanism.³⁵⁸ Vaughan's unorthodox division, however, highlighted the premium food allergists placed on clinical observation, as opposed to prevailing theories of allergy. As the discipline of allergy strove to gain more respect from the medical community, the food allergists' emphasis on inductive reasoning, drawing conclusions on the basis of abundant clinical encounters, fell into disfavour. In 1961, for example, Francis C. Lowell (1909-1980), a past president of the AAA and editor of the *Journal of Allergy*, singled out food allergists in an article on the editorial standards for

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papers relying on clinical evidence. According to Lowell, the observations of food allergists were not a sufficient basis from which to advance the state of allergy; before allergists arrived at any conclusions, ‘such impressions should be put to the test by well thought out experiment’.³⁵⁹

Nevertheless, clinical experiences remained a powerful heuristic for many food allergists. Arthur Coca, whose early career was characterised by academic, rather than clinical, work - he was a professor at Cornell University Medical School from 1910-1932 and the medical director of Lederle Laboratories - began to investigate the diagnostic value of the pulse test in allergy after discovering that his wife’s pulse raced when she ate certain foods.³⁶⁰ He was also motivated by his own health problems, such as severe migraines, dizziness and hypertension, which he attributed to allergy.³⁶¹ Coca echoed Vaughan’s contention that most people suffered at least from minor allergy, believing that Vaughan’s 60 per cent figure was even ‘somewhat conservative’.³⁶² When Coca’s claims about the number of conditions caused by allergy, and especially food allergy, are considered, it is not surprising that he believed Vaughan’s figure to be an underestimate. Coca’s list of food allergy symptoms included symptoms mentioned by many food allergists, conditions such as chronic fatigue, mental disturbance and headaches, but also more provocative symptoms including high blood pressure, baldness, the common cold (one of Coca’s chapters was entitled: ‘You don’t catch your colds - you eat them’), certain types of cancer, multiple sclerosis, glaucoma and even the tribulations of old age. In *The Pulse Test for Allergy*, he mused that the ‘problem of old age will surely change when the new knowledge of food-allergy is put to universal use.

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Instead of planning for the care of the “aged”, we shall have to find the work for them which they will certainly demand in their emancipation from the allergic handicap.³⁶³

Other food allergists were provocative in different ways. Although Theron Randolph’s assertion that food allergies could cause mental health problems mirrored claims made by many food allergists, his contention that one third of his patients were chronically ill due to pollutants in their food, air, water and medication, and that such synthetic chemicals contributed to the ill health of an additional third, was politically, as well as medically, controversial.³⁶⁴ Mackarness, who considered Randolph his greatest influence, similarly asked pointed questions about why allergies and other chronic illnesses had increased so much in the twentieth century. Building on comments made by Rowe in the 1930s, Mackarness believed that allergy, ‘was the greatest cause of illness in Westernized society’ and singled out ‘the industrialized production of food’ as the prime explanation for its rise.³⁶⁵

‘With What We Must Contend’: Reaction to the Claims of Food Allergists

Considering the confident and, at times, spectacular claims posited by many food allergists, it is understandable that food allergy attracted a substantial amount of criticism. In a review of Rowe’s *Elimination Diets and Patients’ Allergies*, for example, Leslie M. Gay (1891-1978), who was president of the SSAAC in 1927 and a frequent critic of food allergists, allowed that while the book would be helpful to ‘any physician who is a disciple of Rowe’, he himself did ‘not agree with the opinion that food plays such a major role in allergic diseases’. Suggesting that ‘the tendency toward a broad interpretation of the definition of allergy, accompanied by reports of cases inadequately studied and not followed for a sufficient period of time, has frequently led to undue emphasis upon the prevalence of food allergy’, Gay proceeded to

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suggest that food allergists such as Rowe would be wise to instead consider ‘the psychosomatic side of human behavior’ and its relation to allergy. Indeed, ‘permanent relief is obtained when a thorough study of his home environment and of his many mental problems is made, and when these all-important factors are adjusted’.³⁶⁶ Gay also bemoaned the dearth of psychosomatic factors in his criticism of Vaughan’s *Allergy: Strangest of All Maladies*, contending that the ‘inexperienced or over-enthusiastic allergist confuses the digestive symptoms of a patient, who is nervous and harassed by financial and domestic problems, for gastro-intestinal or “food” allergy’.³⁶⁷

Other critics accused food allergists of haphazardly prescribing elimination diets that did little more than compromise their patients’ nutrition. In an ardent article entitled ‘With What We Must Contend’, an anonymous writer lambasted an ‘allergist’ practising in the northwest of the United States for limiting a three-year-old girl’s diet to ‘whole rice krisps, rye, rice, arrowroot, leaf lettuce with oil and white vinegar, string beans, spinach, banana, pear, apple juice, grape juice, sugar, salt, butter and (small quantities) of lamb and beef’ in an effort to treat her asthma, even though skin tests revealed allergies to cat hair, feathers, silk and dust. The author contended not only that the patient’s symptoms failed to improve on the diet, but also that infractions of the diet did not lead to additional symptoms. The frustration of the author was evident in the question that concluded the article: ‘Why was the child made to follow an unbalanced diet of approximately ten foods for almost two years during which her infections increased in number and intensity? Why?’³⁶⁸ Allergist Fred T. Grogan similarly ‘deplore[d] the practice of handing the parent a long list of positive food reactions for indefinite

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³⁶⁸ It is possible that the author of this article had treated a former patient of Rowe, given the fact that Rowe practiced in Oakland, California, prescribed quite restrictive elimination diets and wrote numerous articles linking respiratory problems and food allergy.

elimination’, although he also admitted that the ‘only logical treatment for food allergy is elimination and avoidance of the specific food for an extended period’.³⁶⁹ Later, Feingold would also face the criticism that his elimination diet prevented children from getting a balanced diet and, in particular, sufficient vitamin C.³⁷⁰

Despite such attacks, the elevated professional status of many food allergists made it difficult to dismiss summarily their claims as the work of cranks. Rowe, for instance, had been president of the AASA in 1929 and was succeeded in 1930 by another food allergist, George Piness. Not only was Vaughan president of the AASA in 1940, but he was also president of the SSAAC in 1938. Equally important, perhaps, was Vaughan’s medical pedigree: his father, Victor C. Vaughan (1851-1929), was the dean of the University of Michigan Medical School between 1891 and 1920, served as president of the AMA (1914-1915) and was a leading medical figure during the Progressive Era in the United States.³⁷¹ Most of Vaughan’s siblings and some of his children were also prominent physicians. Coca was also a president of the SSAAC (1931), but more significantly founded the *Journal of Immunology* and edited it for 32 years. In conjunction with pioneering allergist Robert A. Cooke (1880-1960), Coca coined the term atopy, a tendency to inherited acute hypersensitivity, in 1923.³⁷²

The food allergists who made seemingly outrageous claims about food allergy were thus well-respected leaders within the discipline. As such, not only did their research get published in leading allergy journals, but it also found support along with criticism. Similar circumstances were present when Feingold published *Why Your Child is Hyperactive*.

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³⁷² Coca and Cooke died within a year of each other in 1959 and 1960, respectively. Their obituaries in the same volume of the *Journal of Allergy* reflected the fact that while Cooke’s reputation as a founder of American allergy was secure, Coca’s work on food allergy and the pulse test had compromised his reputation. Perhaps attempting to sound gracious the author reflected on Coca’s later work by stating that some ‘of his later views were not generally accepted, although their originality and his integrity in presenting them were recognized’.

Conners has suggested that Feingold's strong reputation as a clinical allergist encouraged the medical community to be sympathetic toward his hypothesis when he began reporting it in the early 1970s. Specifically, 'the weight of his authority at first caused Feingold's theory to be taken seriously by scientists. He had already made some fundamental discoveries in allergy and had written a well-regarded textbook of pediatric allergy.'³⁷³ While such support evaporated quickly, it nevertheless demonstrated how an allergist's professional status could affect the reception of his/her claims.

Given the charged atmosphere surrounding food allergy, the claims of its proponents and the standing of many food allergists, it is not surprising that, by 1954, Boston allergists Irving W. Schiller and Francis C. Lowell were acknowledging that 'controversy rages around the clinical importance and frequency of food allergy in a more lively manner than around any other subject in the field of allergy'.³⁷⁴ Although the enthusiasm of food allergists helps to explain such controversy, the nature of food allergy itself, as well as how to diagnose and treat it, also precipitated divergent opinions on the subject. New York City allergist and 1947 AAA president Will C. Spain, in a review of Randolph, Rinkel and Zeller's *Food Allergy*, opined that one chief problem was the inherent difficulty in identifying, diagnosing and treating food allergy:

Of all the problems in clinical sensitization which face the investigator, that of food allergy is the most difficult to resolve. There are three potent reasons for this: first, the patient lacks objectivity in presenting his problem because of his whims, fancies, and aversions relating to various viands, ideas which are often construed by him as proofs of specific food allergy; second, the physician, shorn in at least half of his cases of the benefit of positive food reactions by skin test, tends to be influenced unduly by the description made by the patient of his untoward behavior with certain comestibles; and third, thanks to the ability of

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food allergy to mimic many other nonallergic complaints, the actual allergic nature of the particular problem remains debatable and unsettled.³⁷⁵

Although Spain was generally sympathetic to the authors' approach to food allergy, he concluded his review by warning that:

The methods of diagnosis [the authors] advocate are time-consuming, tedious, and complicated. But so is the condition of food allergy complicated. Anyone in search of a simple and easy diagnostic procedure for this clinical form of sensitization will not find it in this volume nor elsewhere. The subject does not lend itself to an easy solution.³⁷⁶

The ways in which food allergists responded to the difficulties Spain identified contributed significantly to why food allergy was such a controversial topic. As Spain noted in his first point, when dealing with suspected food allergies, allergists were more reliant on the testimonials of their patients than when dealing with other sorts of allergy, such as those caused by pollen, animal dander or dust mites. In the introduction to his textbook, Feingold similarly mentioned the demands and inconveniences food allergy sufferers faced by having to complete comprehensive food diaries and eliminating ubiquitous foods from their diet.³⁷⁷ Rowe, in contrast, described the relationship more positively, stating that 'the absolute determination of all the allergenic causes of many allergic manifestations requires the intelligent and understanding cooperation and analysis of the patient.'³⁷⁸

Reliance on patient accounts was due largely to the unreliability of skin-tests as a diagnostic tool for identifying food allergies, as Spain indicated in his second point. With most allergies, skin-testing was a quick, accurate and relatively safe way to determine which substances were responsible for causing allergic reactions in patients. The procedure,

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³⁷⁸ Vaughan also stressed that intelligent cooperation with patients was essential to the diagnosis and treatment of allergy and, accordingly, wrote two primers geared towards educating patients about allergy: Vaughan, *Allergy*; Warren Taylor Vaughan, *Primer of Allergy: A Guidebook for Those Who Must Find Their Way Through the Mazes of this Strange and Tantalizing State* (St. Louis: C. V. Mosby Company, 1954).

pioneered by Clemens von Pirquet in 1907 to test for reactions to tuberculin, an antigen used in inoculations for tuberculosis, involved introducing a small amount of the potential allergen just below the skin and then waiting to see if the skin reacted by erupting in a wheal.³⁷⁹ It soon became the primary means by which allergists identified most allergens and assessed their severity: the more potent the allergen, the larger the wheal.

In the 1920s, allergists were inclined to believe that food allergies could be identified effectively by employing skin tests. Writing in 1921, Arthur F. Hurst depicted the skin test as being the routine way in which to diagnose food allergy-induced asthma.³⁸⁰ In a 1922 article on the neurological manifestations of food allergy in children, Shannon also described using skin tests successfully to determine the foods to which his patients were allergic.³⁸¹ Three years later Piness and Miller advised their fellow allergists to employ such tests in determining which foods were causing allergies and, therefore, which foods to eliminate from the patient's diet.³⁸² Within the next few decades, however, allergists began to question the effectiveness of skin tests for detecting food allergy. According to well-known gastroenterologist and medical columnist Walter C. Alvarez (1884-1978), 'even the allergists admit that *in cases of food sensitiveness* skin tests are unreliable and only occasionally give a helpful hint'.³⁸³ Prominent food allergists such as Rowe, Randolph, Rinkel and Coca all rejected the use of skin tests for food allergy during the 1930s, 1940s and 1950s, and developed alternative methods for diagnosing food allergy.³⁸⁴ Such tests not only failed to

³⁷⁹ Conjunctival testing, which is done by dropping a small amount of allergen into the eye of the patient, was also a method employed by allergists. This form of testing for allergies is still employed today, but is typically restricted to allergies affecting the eyes that cannot be identified using skin tests.

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³⁸³ Italics in original.

³⁸⁴ While Rowe and Randolph recommended the use of elimination diets to test for provocative foods, Coca believed that allergens caused an increase in pulse rate and, therefore, developed the controversial pulse test for allergy. Rowe and Rowe, Jr., *Food Allergy*; Rowe, *Elimination Diets*;

identify problematic foods, but in some cases, ‘the skin reacts to substances never ingested or to ingestants which provoke no clinical symptoms’.³⁸⁵ Clinical allergists, including Feingold, also expressed concern that intra-dermal testing using food extracts, especially extracts of ‘egg white, buckwheat, nuts, shellfish and fish’, was dangerous and could cause violent, even fatal, reactions.³⁸⁶

Without the evidence of skin tests, food allergists had to rely on the recollections of their patients, as well as the accumulation of their own clinical experiences, in assessing whether food allergies were indeed causing their symptoms and determining which foods were at fault. The lack of more objective diagnostic procedures led to scepticism from both allergists and other physicians about the claims food allergists made regarding the scale of food allergy, as Spain indicated in his final point about the challenges of food allergy.³⁸⁷ New York paediatrician Walter R. Kessler, for example, believed that food allergists were too willing to accept the stories of patients at face value. Complaining that he was ‘constantly confronted by infants whose parents consider their behavior as being outside the realm of normal, and where the diagnosis of “allergy to some food” has already been made by the parent prior to consultation with the physician’, Kessler asserted that such cases were often not examples of allergy, but rather of ‘food *intolerance*’.³⁸⁸ Food allergists, he argued, were all too willing to use ‘food allergy as a “scrap-basket diagnosis” for a variety of problems, for which no other diagnoses have been found’.³⁸⁹

The root of the problem for Kessler, which related to the ineffectiveness of skin tests, was that ‘in the majority of instances [of food allergy] ... it has not been possible to produce

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³⁸⁸ Italics in original.

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objective evidence for the existence of an antigen-antibody reaction and such a mechanism has been assumed rather than demonstrated'.³⁹⁰ In other words, if the antigen-antibody reaction could not be demonstrated, via the use of a skin test, for example, then the patient was suffering from food intolerance, not food allergy. Texas paediatrician Edward L. Pratt, who would later serve as a president of the American Pediatrics Society, elaborated on the difference between intolerance and allergy, stating that:

it is highly important to discriminate untoward reactions from eating a food, a complex mixture with its variable emotional connotations, from reactions following the ingestion of a rigidly defined, specific substance. In everyday practice this difference may sometimes be irrelevant, but to those interested in the role of allergy in Medicine, the distinction is vital.³⁹¹

Essentially, the difference between paediatricians Pratt and Kessler and most food allergists amounted to contrasting definitions of allergy. Kessler believed that 'instances of true food allergy do occur', but insisted that in such cases allergists should be able 'to demonstrate an immunologic reaction ... [that] serves to confirm, and give objective evidence for, allergy as the underlying mechanism' by testing the skin with aqueous extracts of the suspected food protein.³⁹² Although Kessler admitted elsewhere that it was not always possible to demonstrate such immunological mechanisms in food allergy, he nonetheless restricted his definition of allergy to cases in which such a mechanism could be clearly shown.³⁹³ Pratt similarly preferred a narrower sense of allergy and contended that von Pirquet's definition of allergy, which many food allergists employed, 'could encompass practically any disturbance,

³⁹⁰ Manchester paediatrician T. J. David, who also downplayed the link between food additives and behaviour problems, suggested in 1993 that it 'is a paediatric maxim that parents are usually right, but on the subject of food intolerance this is not always so'. Statements such as this one highlight the highly emotive and exceptional nature of food allergy and beg the question of why food allergy is interpreted differently than other ailments.

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including those in interpersonal relations. For example, there would be more fact than humor in the saying, “He is allergic to his mother-in-law.”³⁹⁴

It is clear from *Why Your Child is Hyperactive* and *Introduction to Clinical Allergy* that Feingold would have preferred Kessler and Pratt’s definitions of allergy to that of food allergists such as Rowe, Randolph and Coca. In a telling passage, Feingold stated that:

Allergy is concerned with the defense processes of the body, but the term has wandered far astray from its scientific interpretation. In common speech, allergy has now become synonymous with any form of intolerance or dislike. ‘Allergic’ is frequently used when an individual doesn’t care for a food, though he may tolerate it easily. ‘I’m allergic to onions, plastic pillows and Sam’ usually means that the person simply detests onions, plastic pillows and poor Sam. Even in medicine, the general practitioner or the specialist outside the allergy field sometimes forgets that everything that looks or acts like allergy may not be allergy.³⁹⁵

Feingold also criticised food allergists’ ‘failure to appreciate that nonimmunologic as well as immunologic mechanisms operate to produce unfavourable food responses’ and that this failure helped to explain ‘the great variety of symptoms and diseases which are arbitrarily attributed to allergic mechanisms, often without supporting evidence’.³⁹⁶ Feingold’s words echoed those of Pratt and Kessler, suggesting that the true meaning of the term allergy had been obscured and, more specifically, that many supposed instances of food allergy were merely food intolerance. In particular, Feingold argued that enzymatic deficiencies, chemical irritation, toxic reactions to tainted foods, bacterial contamination and food additives were all examples of non-immunologic adverse food reactions.³⁹⁷ Given that his conception of allergy resembled that of two paediatricians, it is ironic that Feingold, who originally trained as a paediatrician, inferred that it was not allergists, but practitioners outside of the speciality who were responsible for applying the term allergy too liberally. In making this statement

Feingold, whether purposefully or not, ignored the fact that debate over the definition of

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allergy was ongoing within the speciality, and that, more often than not, it was allergists who were more inclined to expand the term.

Food allergy not only made definitions of allergy and methods for diagnosing allergy complicated, but also required a different form of treatment than most other allergies. Skin tests were helpful diagnostic tools, but they also provided allergists with the necessary information to proceed with desensitisation therapy, otherwise known as immunotherapy or inoculation therapy, which would become for allergists during mid-century ‘the cornerstone of their allergy practice’.³⁹⁸ Desensitisation emerged in the early twentieth century in Britain and the United States as an offshoot of vaccine therapy, the use of weakened strains of bacteria to treat active infections such as typhoid, cholera and tuberculosis. Treatment involved inoculating patients over time with small, but increasing, amounts of allergen extract in order to build up their tolerance to allergens such as hay fever-inducing pollen.³⁹⁹

Although Mark Jackson has cited the 1908 instance of British physician Alfred T. Schofield successfully using desensitisation to treat an egg allergy in a 13-year-old boy, most allergists were unconvinced of its efficacy in treating food allergies.⁴⁰⁰ Shannon’s 1922 account of eight food allergy cases, for example, mentioned only the use of elimination diets as a form of treatment.⁴⁰¹ More specifically, Piness and Miller, writing in 1925, advised that: ‘Exclusion of foods has been found far more advisable than attempted immunization, simple abstinence over a varying length of time having been found to produce an immunity.’⁴⁰² Others, including Andresen and Rowe, downplayed the effectiveness of desensitisation in cases of food allergy, while many other accounts failed to mention it as a possible treatment

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altogether.⁴⁰³ Indeed, Vaughan suggested that complete abstinence was more effective in cases of food allergy than desensitisation and that ‘provided the patient avoids the offending allergen sufficiently long [on average, eighteen months], he gradually loses his sensitization and can finally ingest the offending allergen without reaction’.⁴⁰⁴

The inability of food allergists to use desensitisation as a reliable treatment for food allergy served as another indication that food allergy, and food allergists, were somewhat distinct from mainstream allergy. Desensitisation was based in immunological theory and linked both conceptually and historically to vaccination and the notion that immunity could be built up by repeated doses of a weakened pathogen. In the case of allergy and desensitisation, tolerance could be built up by repeated injections of minute amounts of allergen. The problems inherent in desensitising food allergy sufferers, therefore, served as yet another indication that food allergy was not an immunologic process according to the orthodox definition employed by many allergists, including Feingold. Moreover, the idea behind desensitisation was that it was the idiosyncratic patient, not the patient’s environment, that should be altered. Allergy sufferers might attempt to change their environment, ridding it of troublesome dust or pet hair, but they were effectively the passive recipient of the desensitisation treatment provided by the allergist. Food allergists, on the other hand, provided expert knowledge to their patients, but it was the patient who was ultimately responsible for purchasing, preparing and eating the foods recommended by the allergist, not to mention avoiding foods that were banned. Similarly, food allergists believed that it was the environment, not the patient, which had to be changed in the course of treatment. While environmental change meant rigorous elimination diets for food allergists such as Rowe,

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supporters of clinical ecology, such as Randolph and Mackarness, envisioned environmental change as a more comprehensive process that involved changes to how food was grown, produced and preserved, as well as which foods were eaten and how often.⁴⁰⁵

The ineffectiveness of desensitisation treatment for food allergy alienated food allergists from other allergists in another way; it meant that they were somewhat removed from what Jackson has called the ‘global economy of allergy’.⁴⁰⁶ Desensitisation meant not only the provision of a medical service, it also meant the development and provision of a product, namely extracts of various allergens. Gregg Mitman has described how American pharmaceutical companies were quick to exploit the discoveries of British allergists Leonard Noon (1877-1913) and John Freeman regarding desensitisation during the 1910s, and undertook extensive pollen surveys across the United States to identify the most problematic plant allergens in specific regions.⁴⁰⁷ Feingold’s desire to produce an extract of flea saliva allergen suitable for desensitisation is another example of the process’s economic importance in that it led to his securing of a major grant from the NIH and the founding of Kaiser Permanente’s Laboratory of Medical Entomology.⁴⁰⁸ Desensitisation, as well as other treatments for allergies such as antihistamine products, corticosteroids and bronchodilators, none of which were used by food allergists, linked the work of allergists with the research, marketing and sales activities of major pharmaceutical companies.⁴⁰⁹ Although allergists may

⁴⁰⁵ Anthropologist Emily Martin and biophysicist Richard A. Cone have argued that the globalisation of food supply have contributed to both allergy and auto-immune diseases, such as diabetes, multiple sclerosis and arthritis, in both the industrialised and developing world. They argue that, when people eat locally-grown food and whole, unprocessed, food, they inadvertently ingest particles of potential allergens, such as pollen, which then desensitises them to such allergens. Moreover, they argue that pesticides, preservatives and toxic pollutants act as ‘adjuvants’ which attach to food molecules, making them allergenic. For Cone and Martin immunological disease has a strong environmental dimension that is tied into the global food economy.

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have debated, for example, whether corticosteroids were more effective than desensitisation, the sale of either was an example of the symbiotic relationship that existed between the allergist and the pharmacist.⁴¹⁰

Food allergists, on the other hand, enjoyed no such relationship with pharmaceutical companies. Rather than involving the purchase of a medical product, the elimination diets that food allergists prescribed restricted what patients could purchase and encouraged them to shop more cautiously. This was especially the case for Randolph's patients who, as early as the 1950s, were encouraged to purchase un-processed, organic and pesticide-free foods.⁴¹¹ Writing with science writer Ralph Moss in 1980, Randolph admitted that to 'name corn, wheat, milk, eggs, beet and cane sugar as the sources of illness, even in a minority of the population, will not make many friends among the commercial producers of these foods'.⁴¹² As will be demonstrated below, Feingold's warnings about food additives disturbed not only the food processing industry, but also the pharmaceutical industry in that his solution to hyperactivity eliminated much of the need for drugs such as Ritalin. Moreover, the tendency for food allergists such as Rowe, Coca and Vaughan to claim that food allergy caused a great number of ailments misdiagnosed as other types of allergy, infections or psychiatric problems meant that food allergists diverted patients from the products recommended by orthodox allergists and developed by pharmaceutical companies and other industries to treat such complaints. There were speciality foodstuffs developed and marketed for people who were allergic to staples such as milk, egg and wheat, for example, Ditek Oat Crisps, biscuits free of egg, milk, wheat, corn and barley, but food allergists very rarely mentioned such products.

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Indeed, food allergists such as Randolph instead appealed to food producers to provide clear, comprehensive and accurate lists of ingredients on their product labels, a request that placed greater demands on industry with little or no benefit in return.⁴¹³

Conclusion

In many ways food allergy can be seen as a distinct and controversial offshoot of allergy. Proponents of food allergy employed a much broader definition of allergy than that stressed by orthodox allergists, and made bold claims about how many people were affected by ingested allergens. Since skin testing for food allergy was ineffective, food allergists had to rely on the testimonials of their patients and their own observations, measures that may have been useful clinically, but that struck many other allergists as un-scientific and too open to interpretation. Food allergy was also difficult to treat and relied not so much on the skill of the allergist, but on the willingness and determination of patients to adhere to often restrictive elimination diets. The use of such diets also precluded food allergists from intersecting their work and research with that of pharmaceutical companies whose allergen extracts, antihistamines and, by the 1970s, bronchial inhalers were part and parcel of the orthodox allergist's armamentarium.

Opinions on food allergy were still divided by the time *Why Your Child is Hyperactive* was published in 1974. A 1975 issue of *Pediatric Clinics of North America*, for example, featured an article by William Crook in which food allergy was dubbed the 'Great Masquerader', and

⁴¹³ This has changed somewhat in the last decade with the increased awareness of acute peanut allergy and the high profile given to fatal cases. A recent television advertisement campaign in Canada for Mars Bars featured the company's claim that its chocolate bars were produced in a facility that was a peanut-free zone. Nevertheless, the cost of labelling products that might come in contact with nuts during production, as well as the attempted elimination of nuts from certain production facilities, must make it difficult for companies to derive any tangible benefit from the increase in peanut allergy diagnoses.

blamed for hyperactivity, learning difficulties, headache, enuresis, gastrointestinal complaints and respiratory problems.⁴¹⁴ The same issue, however, also published a commentary on food allergy by another American allergist, Charles D. May (d. 1992), in which he warned that

In the absence of means for rigorous identification of the immunologic mechanisms, uncritical claims of relations of foods to symptoms can be expected, and unsupported ‘systems’ of diagnosis and treatment will flourish. Those who resort to dubious practices will surely be shunned by adherents of the scientific method in clinical medicine. The afflicted and the uncritical will join in creating another quackery by resorting to some ‘system’ as a crutch to hobble along with until better means of relief can be found. Rather than calling food ‘allergy’ the ‘Great Masquerader’ among causes of a long list of subjective complaints (e.g., tension-fatigue), common to those overwhelmed with the trials and tribulations of life, this use of food ‘allergy’ may be recognized as the Current Crutch. Such has been the story of quackery, and so it will always be until the last gaps in our knowledge are filled.⁴¹⁵

Six decades after B. Raymond Hoobler cautiously suggested that food allergy might be the cause of common chronic complaints, therefore, allergists in the 1970s were undecided as to whether food allergy was the ‘Great Masquerader’ or a form of ‘quackery’.⁴¹⁶

It was within this divisive context that Feingold had to decide whether or not to link his theory about hyperactivity with those of food allergists such as Crook, or with those of more orthodox allergists, such as May. It is also within this context that Feingold’s decision to situate the origins of his diet outside of the history of food allergy must be understood. It is clear that, on the surface, Feingold’s hyperactivity hypothesis fitted neatly into the history of food allergy and, especially, the tradition of linking food allergy with psychological problems including hyperactivity. If Feingold had re-considered whether or not the reactions he observed were allergic, or even refused to bother with such questions, as food allergists such

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⁴¹⁶ There were some physicians, such as Memphis paediatrician, Fred T. Grogan, whose opinions regarding food allergy were more balanced or, perhaps, less dogmatic. Prominent British allergist, A. W. Frankland, who sympathised with the work done by Albert Rowe and brought him to Britain for a lecturing tour, also adopted a more flexible perspective to food allergy. On the whole, however, it appears that by the 1970s the issue of food allergy tended to split physicians into opposing camps. Grogan, ‘Food Allergy’; , Interview.

as Crook did, he would have probably been seen as part of the tradition started by Shannon, Piness and Rowe and carried on by Crook, Randolph and Mackarness.⁴¹⁷

It is also apparent, however, that the theories, diagnostic techniques and treatment strategies promoted by food allergists alienated them from their orthodox colleagues. The result of this alienation was the creation of a sub-field of sorts, removed from the remit of mainstream allergy and without many of the theoretical, political and economic commonalities that might have allowed allergists to work within both paradigms. As such, associating with food allergists meant severing ties with the mainstream allergy community and losing a great deal of respect as well. Indeed, this was the misfortune of Arthur Coca, among the most important allergists of the inter-war period, whose reputation suffered considerably when he began theorising about food allergy late in his career.⁴¹⁸ Although in retrospect the historian might see many similarities in the career trajectories of Coca and Feingold, it seems clear that Feingold's intention was to avoid Coca's fate by emphasising his traditional background and orthodox beliefs about allergy. From this perspective, Feingold's choice to shun food allergists and attempt to retain his ties to traditional allergy appeared to make some strategic sense.

Feingold's decision to describe the origins of his diet as distinct from the history of food allergy was not, however, merely strategic. The articles and books Feingold wrote on bronchial allergic disease, flea bite allergies, psychosomatic factors in allergy and clinical allergy reflected the views of an orthodox allergist and the restricted definition of allergy that such a perspective presupposed. Feingold's traditional outlook predisposed him to interpret

⁴¹⁷ Doris J. Rapp, a paediatrician who promoted her own allergic theory of hyperactivity during the late 1970s, was more willing than Feingold to show how her theories were linked to those of earlier food allergists. Rapp mentioned that she was 'ashamed to admit that from 1960 to 1975 while in practice as a pediatric-allergist, I seldom recognized or diagnosed this problem. Then, as often happens in medicine, my patients taught me.' Doris J. Rapp, *Allergies and the Hyperactive Child* (New York: Simon and Schuster, Inc., 1979), 3-12.

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his observations of the effects of food additives on children's behaviour in a markedly different manner than the food allergists who had been claiming for decades that food allergies could cause mental health problems. Ultimately, it might have been more surprising for Feingold to have abandoned the allergy paradigm within which he had operated successfully for decades and embrace the radically different one espoused by food allergists.

The reception of Feingold's diet by the medical profession will be the subject of Chapter 9. At this point, however, it is sufficient to state that, despite Feingold's efforts to associate his hyperactivity hypothesis with contemporary research in allergy and immunology, most allergists downplayed his findings and, as a whole, his thesis was vetted more often in psychology and psychiatry journals, rather than in those dedicated to allergy and immunology. Perhaps this was an unexpected result of Feingold himself emphasising the non-allergic aspect of the food additive reaction. It is likely that, despite Feingold's careful elucidation of the origins of his diet, most allergists saw his work as an extension of similar observations made by food allergists for sixty years and, thus, dismissed it fairly rapidly or ignored it altogether. Given his efforts to avoid this fate, Feingold was understandably hurt by this reception. According to an anonymous associate:

Dr. Feingold was somewhat naïve [and] thought that he would be applauded as a benefactor of human well-being. I think that he was shocked by the rejection and criticism. From my viewpoint, he had been a traditional allergist who veered off the conventional path, and experienced what all iconoclasts experience: they are ignored, dismissed, scorned or ridiculed.⁴¹⁹

Feingold's naïveté may indeed have inflated his sense of how palatable his theory would be to his colleagues in allergy. He also expressed to colleagues that his peers did not fully understand his hypothesis, describing how he was 'confident they do not appreciate the complexity of the problem'.⁴²⁰ Other associates, especially those who worked with him at

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⁴²⁰ Letter from Ben. F. Feingold to Beatrice Trum Hunter, 13 April 1979, Beatrice Trum Hunter Collection, Box 47.

Kaiser Permanente, believed that other aspects of his personality contributed to his misjudgement. Alice Friedman, who assisted Feingold during the 1960s, described him as ‘an extraordinarily autocratic gent’ who did not suffer his critics particularly well.⁴²¹ Friedman also suggested that Feingold tended to take sole credit for accomplishments and discoveries that should have been shared amongst others, stating that, according to Feingold: ‘No matter what anybody else does, it’s the chief of the clinic who gets the credit for it ... It’s always the chief.’⁴²² Friedman has claimed that she was responsible for alerting Feingold to the diet, designed by Stephen Lockey at the Mayo Clinic, which served as the model for the Feingold diet.⁴²³ This being the case, Feingold might also have been loath to share credit with food allergists for the notion that nutrition could affect behaviour.

Dov Michaeli, one of Feingold’s researchers during his flea bite allergy research, described Feingold as being ‘despotic’ and ‘a bit full of himself’, although he himself got along well with Feingold, partly because Feingold was not his direct supervisor and they tended only to see each other socially.⁴²⁴ According to Michaeli and others, however, Feingold had disagreements with many of his other colleagues, most notably many of the food allergists and clinical ecologists who might have sympathised with his ideas about hyperactivity. While Michaeli stated that Feingold knew of Rowe, who worked across the San Francisco Bay in Oakland, he only mentioned him ‘in a denigrating way’.⁴²⁵ Environmental toxicologist Bernard Weiss, who enjoyed a professional relationship with Feingold, also declared that Feingold ‘despised’ food allergists and clinical ecologists such as Rowe, Randolph and Mackarness as well as the work that they did.⁴²⁶

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⁴²⁶ Bernard Weiss, Interview.

The picture painted by Feingold's associates is not a particularly endearing one and far removed from the charismatic, grandfatherly persona shown to the public when he was promoting his diet. Feingold is presented in these testimonials as a rigid, egotistical autocrat whose contempt for food allergy and thirst for scientific acclaim clouded his judgement. That said, the choices Feingold had before him upon writing *Why Your Child is Hyperactive* were not easy ones. In many ways he was caught between his observations of hyperactive children and his long-held perceptions about food allergy and its proponents, perceptions shared by most of his colleagues. The compromise reached by Feingold in his depiction of his theory's origins, therefore, can be interpreted as an attempt to reconcile a conflict between his clinical impressions and his theoretical underpinnings as a traditional allergist. As the next section demonstrates, this seemingly irreconcilable gap between theory and practice and between laboratory and clinic would remain a key feature of debates about the Feingold diet and helps to explain how and why his hypothesis became so popular.

Part II

The Dissemination of the Feingold Diet

Chapter 5

From *JAMA* to the *National Inquirer*: The Popularisation of the Feingold Diet

The previous section on the origins of the Feingold diet highlighted how the contentious nature of food allergy and clinical ecology made it difficult for Feingold to contextualise the origins of his hypothesis in a way that would appeal to both unorthodox and traditional allergists. Despite developing a theory of hyperactivity that appeared to connect with the history of food allergy dating back to the early twentieth century, Feingold chose to distance his theory from this history and, instead, associate his hypothesis with those of more respectable and appreciated researchers. Feingold's efforts in this respect underlined his background as a well-respected and orthodox allergist who had published regularly on numerous subjects in reputable medical journals, operated a lucrative allergy practice in Los Angeles and founded and managed a dozen allergy clinics for Kaiser Permanente in northern California. His attempt to explain the origins of his hypothesis in a rational, plausible and scientifically-sound manner exemplified his desire to earn the approbation of like-minded physicians, rather than that of controversial food allergists and clinical ecologists.

Given Feingold's desire that his theory gain medical respectability, it may seem strange that he chose to disseminate his idea not through articles in leading medical journals, such as *JAMA*, *AJP* or *Pediatrics*, but in a popular book aimed at parents and published by Random House. Prior to his work on hyperactivity, Feingold was well-acquainted with publishing his research findings in medical journals. Feingold's flea bite allergy research in the 1960s, for example, was accompanied by ten articles he wrote or co-authored in scientific journals ranging from *Experimental Parasitology* to the *Journal of Immunology*.⁴²⁷ Feingold's first

⁴²⁷ B. W. Hudson, Ben F. Feingold, and L. Kartman, 'Allergy to Flea Bites I: Experimental Induction of Flea-Bite Sensitivity in Guinea Pigs', *Experimental Parasitology* 9 (1960), 18-24; B. W. Hudson, Ben F. Feingold,

observations about the allergenic effects of food additives were also published in a medical journal, the *Annals of Allergy*, in 1968.⁴²⁸ Moreover, Feingold was keen to submit his hypothesis about food additives and hyperactivity to the scrutiny of his peers, and proceeded to do so at the allergy section of the 1973 meeting of the AMA in New York. Why, then, did Feingold compromise the respectability of his theory, not to mention that of himself, by publishing it in *Why Your Child is Hyperactive*? Furthermore, what was the impact of this decision on how physicians and parents received and understood his theory?

This section will address these questions by examining the process by which word about the Feingold diet spread. In this opening chapter I will argue that Feingold's decision to write for a popular audience was not entirely his own and, in many ways, was a reaction to circumstances thrust upon him during the period between 1973 and 1975 by an ambivalent medical community, by parents of hyperactive children who were frustrated with current explanations and treatments for hyperactivity, and by a media that voiced alarm about the safety of the food supply, ecological issues and the manner in which mainstream physicians were treating hyperactive children. While the discomfort exhibited by certain members of the medical community about Feingold's line of inquiry made it difficult for him to publish his hypothesis in leading medical journals, the public's parallel concerns about hyperactivity and the environment meant that the media was eager to investigate his claims and publish stories about them in newspapers and magazines. Correspondingly, the populism that Feingold eventually embraced greatly affected the reception of his theory by the medical community, the media and the public. Although physicians were largely unimpressed by

and L. Kartman, 'Allergy to Flea Bites II: Investigations of Flea Bite Sensitivity in Humans', *Experimental Parasitology* 9 (1960), 264-70;;

Feingold's decision to write a popular book, instead of publishing his ideas in a series of academic articles, and were unmoved by, if not resentful of, his emerging celebrity, parents and the media were captivated. *Why Your Child is Hyperactive* introduced Feingold's ideas to millions of parents, not only through book sales, but also through television and radio interviews and the hundreds of newspaper and magazine articles that discussed his new theory during the 1970s.

Although *Why Your Child is Hyperactive* was written with physicians in mind, it was clearly a popular book published by a major publisher for a popular audience consisting largely of parents. By the time of its publication, it was evident to Feingold that the path to legitimising his hypothesis about hyperactivity did not necessarily involve running the gauntlet of the medical approval process, consisting of double-blind trials and peer-reviewed articles, but instead meant connecting with families via newspaper articles, television interviews, the fostering of Feingold Associations and even individual consultations and telephone conversations with parents. Furthermore, Feingold would eventually exhibit little discrimination with regards to which newspapers and magazines he would give interviews; parents would eventually read about the Feingold diet not only in respected newspapers such as the *New York Times* and the *Washington Post*, but also tabloids such as the *National Inquirer* and even the pornographic men's magazine, *Penthouse*.⁴²⁹ While this chapter examines how such a development took place by tracing Feingold's attempts to convince the medical community that food additives could cause hyperactivity, the two that follow turn to

⁴²⁹ According to Jan Hersey, Feingold 'used whatever techniques he could use to reach the parents. And I remember one time somebody said that there was an interview of him in the *National Inquirer* and I was shocked and that I realised that he would never interview them or use them as a resource, but he just wanted to talk to anybody who could get the information out; he was very passionate about wanting to reach the children.' Jane Hersey, Telephone Interview, 15 August 2007. Another Feingold parent, Lora Hollins of Michigan, related how she learned about the Feingold diet from an article in *Penthouse*. Lora Hollins, Telephone Interview, 17 February, 2008.

why Feingold's concerns about food additives and hyperactivity resonated so strongly with the American public.

Mixed Messages from the AMA

One of the striking aspects of how knowledge of the Feingold diet spread across the United States and beyond is the speed with which this process took place. Feingold began treating hyperactive children in San Francisco with his elimination diet in the middle of 1972, and by the autumn of that year he had prescribed the diet to a mere twenty-five children, only fifteen of whom experienced improvements in their behaviour.⁴³⁰ By the autumn of 1973, however, Feingold had reported his clinical findings about hyperactivity and food additives to the AMA and the Royal Institute in London, spoken to the international media about his hypothesis, had the manuscript of his London presentation submitted into the Congressional Record and was the subject of numerous magazine and newspaper articles.⁴³¹ The explosion of interest in 1973 was not so much due to Feingold's active promotion of himself or his diet, however, but rather to the high degree of receptivity that the public and, initially, the medical community exhibited towards his theory.

Although Feingold would become a willing and eager participant in such publicity and media attention, it does not appear evident that, during this first year of prescribing the Feingold diet, he was the instigator of it. In the words of Jane Hersey, director of FAUS, 'Feingold did not go out and beat the bushes to become famous; that was not what he was doing. Once the genie was out of the bottle you couldn't put it back again.'⁴³² Instead, Feingold seems to have proceeded rather cautiously with regards to his thesis, and attempted to gain the support of his medical peers prior to making conclusive claims. Feingold's caution is reflected in the

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language used by himself and others to describe how his fellow physicians, the AMA, the American Congress and the media learned about his hypothesis, and also suggests that Feingold's role in attracting media attention was initially largely passive. In *Why Your Child is Hyperactive*, for example, Feingold stated that by 1972 he had begun discussing his observations about food additives and hyperactivity to 'other doctors and with friends', and that the media in San Francisco were alerted simply because: 'Word got around that I had a theory about the hyperkinetic-learning disabled child.' According to Feingold, the result of such rumours was that by 'late October 1972 I found myself before the cameras of KPIX-TV, San Francisco, discussing what I had learned of the H-LD and talking about my slowly hardening hypothesis'.⁴³³

It is possible, although difficult to substantiate, that this television appearance prompted the AMA to invite Feingold to make a presentation at their June 1973 meeting in New York. Indeed, Feingold recalled being invited to the meeting in late 1972, after his first television appearance occurred.⁴³⁴ That Feingold described his hypothesis as 'slowly hardening' also suggested that he desired the opportunity to test his theory more thoroughly before submitting it to the scrutiny of his peers, let alone the media. Elsewhere in *Why Your Child is Hyperactive*, Feingold mentioned 'casually' discussing his observations in April 1973 with colleagues Alice Friedman and Don German, who had taken over from Feingold as chief of allergy for Kaiser Permanente when Feingold was fighting cancer during the late 1960s. Both physicians were 'skeptical' and thought that the positive responses of children prescribed the elimination diet 'might have been a psychological reaction to the diet program

⁴³³ Although rumours were likely crucial in spreading word about Feingold's theory about hyperactivity, it is also possible that Feingold's publication of *Introduction to Clinical Allergy* in 1973 was also responsible. This is because in this textbook, aimed at both allergists and other physicians, Feingold discussed 'behavioral disturbances in both children and adults which have been attributed to food additives'. Moreover, on the inner jacket of the textbook, Feingold's observations about food additives and disease are highlighted.

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and to the constant attention and vigilance of the parents'. Admitting that he had these suspicions himself, Feingold added that 'As a very conventional medical doctor, I have always been leery of "cure" by diet.'⁴³⁵

Feingold's apprehensiveness spurred him to take advantage of the opportunities provided him in 1973 to discuss his theory with his medical peers. He described these opportunities, specifically his invitation to speak at the allergy section of the June 1973 AMA meeting in New York, as 'an excellent forum for peer evaluation of our observations on behavior and dietary intervention'.⁴³⁶ In other words, Feingold was not so much looking for publicity as a chance for feedback from other allergists and paediatricians. Feingold also insisted that he was never responsible for making overtures to organisations such as the AMA, or, indeed, the media; it was the AMA that invited him to speak at their annual meetings and not the other way around.⁴³⁷ The only organisation Feingold did contact about his theory prior to his AMA presentation was the FDA, to whom he began writing in May 1973, urging that they market more additive-free foods and require fuller and clearer disclosures of additives on food product ingredient labels.⁴³⁸

Feingold's letters to the FDA notwithstanding, reports in the media of his June 1973 presentation to the AMA suggested that the San Francisco allergist still held some reservations about the validity of his theory, and that he desired more time to test it and discuss it with other physicians. An article in the *Hartford Courant*, for example, reported that 'Dr. Ben F. Feingold says he has no solid evidence yet for this suspicion', but also that Feingold believed his 'clinical observations call for a look into what effects on child behavior

⁴³⁵ Feingold's comment about being leery about diet cures might be a veiled reference to Albert Rowe, the pioneering food allergist who operated a lucrative food allergy clinic in Oakland, California, a city just on the other side of San Francisco Bay.

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⁴³⁷ Ibid.

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there may be from various of the thousands of chemicals added to foods'.⁴³⁹ Such sentiments were also reflected in a *Chicago Tribune* article which followed the AMA meeting and reported that, while Feingold was unclear about the physiological process by which additives caused hyperactive behaviour, he believed that his observations warranted thorough investigation.⁴⁴⁰ Moreover, a *Newsweek* article in July 1973 stated that 'because he has not yet done a controlled study, Feingold cautions that his observations must be regarded as preliminary'.⁴⁴¹ Further evidence of Feingold's caution can be found in his 1973 *Introduction to Clinical Allergy* in which he refrained from making definitive statements about food additives and hyperactivity and, instead, simply posed the question: 'Is it possible that some cases of so-called MBD [minimal brain dysfunction] may be manifestations of neuro-physiologic disturbances induced by certain chemicals such as the food additives?', and suggested that further investigations were required to test his hypothesis.⁴⁴² In many of Feingold's other early media interviews he similarly preferred to pose questions such as this, rather than giving definitive comments.⁴⁴³

Although Feingold believed his clinical observations, consisting of only 25 hyperactive patients in June 1973, to be somewhat preliminary and requiring additional study, the AMA was initially more enthusiastic. Such enthusiasm is evident in the fact that, after Feingold submitted his manuscript for his presentation, the AMA asked him if he would be willing to participate in a news conference on 25 June 1973, a day prior to his presentation. Feingold 'accepted and met with between 75 and 80 correspondents from around the world, following

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⁴⁴³ For example, in Morton Mintz's October 1973 story on Feingold's theory, a story which made the front page of the *Washington Post*, Feingold is quoted as asking, but not answering, the questions: "Is it possible to attribute the increase in hyperkinesis and learning difficulty to the increased consumption of these chemicals in our foodstuffs?" [and] "Do the additives ingested by the mother during pregnancy affect the unborn child?" In this story Feingold also mentioned that, since there were other causes of hyperactivity, his diet did not work for all of his patients.

which the report that hyperkinesis responded to a diet eliminating artificial food colors and flavors was covered by practically all the news media'.⁴⁴⁴ Reacting later to such media interest, Feingold stated: 'Since I am not a behaviorist nor a psychiatrist, but rather an allergist and immunologist, I was not prepared for this response.'⁴⁴⁵

The reasons for such media interest will be discussed more fully in Chapters 6 and 7, but suffice it to say that Feingold's emerging theory resonated strongly with contemporary American concern about the food supply, the chemical environment and childhood behaviour problems. Nevertheless, Feingold's initial goal was not to publicise his theory but to seek the approval of the medical community and, as such, he endeavoured in the autumn of 1973 to present his ideas to medical colleagues at scientific conferences and in medical journals.⁴⁴⁶ For instance, Feingold submitted the preliminary findings he presented to the AMA for publication in *JAMA*.⁴⁴⁷ He also accepted an invitation to speak at an international symposium on food in September 1973 at the Royal Institution of Great Britain and, accordingly, submitted another article for publication in the *British Medical Journal*.⁴⁴⁸ During that September, Feingold also agreed to write a signed editorial for the medical journal *Hospital Practice* which appeared in October 1973.⁴⁴⁹ Finally, towards the end of 1973, Feingold accepted a second invitation to speak at the AMA's 1974 meeting, this time in Chicago.⁴⁵⁰

Despite Feingold's overtures to his fellow physicians, the reaction of many influential medical associations during the period 1972 to 1975 was ambivalent. On the one hand,

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associations, such as the AMA and the California Medical Association (CMA), invited Feingold to present his theory to their annual meetings in 1973-1974 and 1975, respectively. The AMA, in particular, scheduled another press conference in 1974 to precede Feingold's presentation and, again, media coverage was heavy, resulting in almost daily stories in American print, radio and television media.⁴⁵¹ On the other hand, the AMA refused to publish Feingold's findings in either 1973 or 1974 and the CMA refused to publish his findings in its journal, then called the *Western Journal of Medicine*, following Feingold's presentation to them in 1975.⁴⁵² Feingold's submission to the *British Medical Journal (BMJ)* was similarly rejected.

The mixed response to Feingold's theory during this early period is perplexing. Why did the AMA promote Feingold's thesis by inviting him to their meeting and arranging large press conferences, only to reject Feingold's submission for *JAMA*? Given the fact that the first clinical trials which tested the Feingold diet did not emerge until 1976, it is clear that the AMA's decision not to publish Feingold's findings was not based upon scientific evidence or the reports of other researchers. Although the reasons for the AMA's reversal are difficult to pinpoint, a number of key factors help to explain why Feingold ultimately abandoned his goal of achieving the sanction of the medical community for his ideas concerning hyperactivity.

⁴⁵¹ For example, Feingold was interviewed in June 1974 on the popular Patricia McCann Magazine on New York's WOR radio. Newspaper articles about the Feingold diet appeared in many major American newspapers in June 1974, including the *New York Times*, the *Chicago Tribune*, the *Hartford Courant* and the *Washington Post*. Feingold, 'A View from the Other Side', 6-7, 11;

⁴⁵² Feingold had been published in both *California Medicine*, one of the predecessors of the *Western Journal of Medicine*, and *JAMA* in 1949 and 1951, respectively. One possible reason that the CMA was reluctant to publish his findings with regards to hyperactivity, however, involved not only the controversial nature of his research, but also the fact that he worked for Kaiser Permanente. According to Feingold's colleague at Kaiser Permanente, Alice Friedman, Feingold had been a member of the CMA when he worked in private practice in Los Angeles, but when he joined Kaiser Permanente in 1951, the San Francisco branch of the CMA 'blackballed him' and prevented him from joining them. This is because Kaiser Permanente provided private medical insurance, a concept that was viewed by many American physicians as being socialist and a threat to their income.

The chief reason why the AMA was initially interested in Feingold's theory seems to have been Feingold's excellent reputation as a paediatric allergist. According to C. Keith Connors, a pioneer in hyperactivity research and key player in the investigations into the Feingold diet, 'the weight of his authority at first caused Feingold's theory to be taken seriously by scientists. He had already made some fundamental discoveries in allergy and had written a well-regarded textbook of pediatric allergy.'⁴⁵³ Even Morris Lipton (1916-1989), the University of North Carolina psychiatrist who headed the food chemical industry's investigation into Feingold's claims, stated in 1977 that 'Dr. Feingold and his work are well known to me, and as a reputable physician I must take him seriously – up to a point.'⁴⁵⁴ Not only was Feingold a leader within the allergy community in California with impressive clinical experience and publication record, but he also sat on the AAA's Committees on Food Allergy and the Committee on Insects and Insect Allergy during the 1960s and 1970s.⁴⁵⁵ Moreover, it has also been proposed that Feingold's antipathy for the clinical ecology movement might have also reassured the AMA that they were inviting a fairly conservative allergist to speak to the media, and not a radical such as Theron Randolph.⁴⁵⁶ On the other hand, nutrition scientist Alex Schauss, who knew Feingold during the 1970s and 1980s, suggested that the AMA re-invited Feingold in 1974 because 'he was controversial and [they] knew it would raise attendance'.⁴⁵⁷

Perhaps it was such controversy, however, that ultimately caused the AMA to distance itself from Feingold and his theory.⁴⁵⁸ Despite Feingold's reputation as a conservative allergist, his

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⁴⁵⁶, Interview.

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⁴⁵⁸ The AMA's history of protecting the reputation of its members from controversy and rooting out quackery is indicative in other ways. The only AMA archive open to historical researchers, for example is its archive on 'Historical Health Fraud and Alternative Medicine' which the AMA describes as 'the nation's finest collection on medical quackery' and the 'result of nearly seventy years of activity [1906-1975] by the AMA's Department of Investigation'. Moreover, the 'fraudulence existing today in matters pertaining to medicine and nutrition' was one of the traditional targets of the AMA's Department of Investigation. American Medical

theory about hyperactivity had profound implications for food, chemical and pharmaceutical companies. As *Business Week* contributor Geraldine Pluenneke described, commenting on the publication of *Why Your Child is Hyperactive*:

a time bomb is ticking beneath the dust wrapper of this book, and it could explode into another widespread controversy over food additives ... Count Chocula and Boo Berry cereals. Decongestants and bear-shaped vitamins. Cavity-tracking toothpastes, self-basting turkeys; corn oil margarines, colas ... a San Francisco allergist is advancing an empirical theory linking the common ingredients in such products -- synthetic colors, flavors, and enhancers -- with a seemingly epidemic rise in learning problems.⁴⁵⁹

Pluenneke went on to state that Feingold's:

hypothesis spells bad press for food men. Some companies are already keeping a low profile while test-marketing new products to avoid provoking attacks by food naturalists who already question the long-term dangers of food chemicals. Feingold's theory has an immediacy that could have an impact at the checkout counter: the promise of a rapid dietary escape from a problem that is now routinely handled with a battery of stimulant, tranquilizing, and antidepressant drugs.⁴⁶⁰

According to Jane Hersey Feingold's previous work on reactions to food additives, as well as similar work conducted by other researchers such as Stephen D. Lockey, Frederic Speer, Guy Settignano and F. H. Chafee on reactions to food dyes such as tartrazine,⁴⁶¹ had already, by the 1970s, caught the attention of food chemical companies. Although there is no direct evidence linking the food chemical industry to the AMA, Hersey and others have suggested that, given the lack of other explanations, there is a likelihood that industry lobbyists helped convince the AMA to stop promoting Feingold:

Here you have an allergist, a doctor who comes up with information which is extremely damaging to huge multi-billion dollar industries. What did the multi-billion dollar industry do when they were threatened? And they were threatened. [laughs] If parents find out that Jell-o and Kool-Aid and you name it, you know, Cocoa Puffs or whatever it is, Trix cereal, if parents find out that these things contain additives that harm children, look at the bottom line there ... Now why would the AMA drop the whole thing when they were the ones who had promoted Dr. Feingold's work and done all of this? I have no idea, but

Association, 'Historical Health Fraud and Alternative Medicine', www.ama-assn.org/ama/no-index/about-ama/17954.shtml, accessed 3 March 2009.

⁴⁵⁹ AAAAI, 'AAAAI Records', Box 242, Folder 10.

⁴⁶⁰ Pluenneke, 'Food Chemicals', 12.

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I do know that all kinds of medical groups, uh, get money from vested interests and they accept it. The American Academy of Pediatrics was given a whole bunch of money from the Soy formula people. Afterwards, they toned down their opposition to soy formula. The American Pediatric Dental Foundation got a huge contribution from Cadbury-Schwepps, the candy people ... The only thing that makes sense to me, although there's no way I can prove it, is that someone paid somebody a lot of money.⁴⁶²

Given that the AMA does not currently allow the vast majority of its records to be viewed by historians, it is impossible to say whether or not Hersey's unfounded speculation has any evidence to support it. It is only apparent that at some point following Feingold's 1973 and 1974 presentations to the AMA a decision was made to stop promoting his diet and prevent his ideas from being published in *JAMA*.⁴⁶³ The decision might not have been influenced by the food chemical industry, but it did parallel initiatives made by the food chemical industry in 1974 and 1975, under the auspices of its research organisation, the Nutrition Foundation, to investigate the Feingold diet and downplay its significance.⁴⁶⁴

The steps taken by the Nutrition Foundation to investigate Feingold's hypothesis included the issuing of a December 1974 report that focussed on his theory and a January 1975 conference on the subject. Despite the fact that the last paragraph of the Nutrition Foundation's proposal stated that 'no publicity will be given to the findings until the Committee has approved the report for release', a week later a preliminary report was published with a number of 'conclusions' about Feingold's theory. Among them were the warnings that no 'controlled studies have demonstrated that hyperkinesis is related to the ingestion of food additives', and that the 'nutritional quality of this diet has not been evaluated and it has not been determined

⁴⁶² Jane Hersey, Telephone Interview. The records of the AAAAI also indicate that it had actively sought out ties with the food industry. One of its proposed projects of its Food Allergy Committee in 1984, for example, was the: 'Creation of formal relationships with our Committee, and the American Academy of Allergy and Immunology, to scientists connected with the food industry.' The rationale behind such a relationship was not to warn the public about possible food allergies, but to reassure them about processed food in restaurants. Box 243, Folder 2.

⁴⁶³ It could also be that the editors of *JAMA* differed with those organising the AMA conference with respect to Feingold's theory and whether it should be published. Thanks to Rima Apple for offering this suggestion.

⁴⁶⁴ The Nutrition Foundation was renamed the International Life Sciences Institute in the 1980s.

if it meets the long-term nutrient needs of children'.⁴⁶⁵ Although these preliminary 'conclusions' were originally intended to stay out of the press, they were given a great deal of exposure in the American media.⁴⁶⁶ Moreover, the American Academy of Pediatrics (AAP) proceeded to print the Nutrition Foundation's statement verbatim in their newsletter, along with the Nutrition Foundation's New York address.⁴⁶⁷ Other medical organisations, including the AAA, also served notice to the public that they were unwilling to endorse Feingold's theory.⁴⁶⁸ The responses of all three of these medical associations to the Feingold diet, not to mention the response of the Nutrition Foundation, occurred well before any controlled studies into the diet emerged. The AMA might have instigated interest in the Feingold diet by organising the press conferences that accompanied Feingold's presentations, but, like other medical associations, they were also quick to distance themselves from his theory when it appeared to be too contentious.

The Popularisation of the Feingold Diet

Ultimately, the reasons that prompted the AMA to reverse their position on Feingold's hypothesis proved to be less important to the fate of the Feingold diet than the impact of this reversal on how Feingold subsequently chose to promote his ideas. Faced with the rejection of his colleagues, but also inundated by queries from parents and media interview requests, and continuing to experience clinical success with his diet, Feingold had to decide how to spread the word about his hypothesis. The decision of the AMA to disregard Feingold's claims was a primary factor in convincing Feingold that the best way to promote his hypothesis was not necessarily by gaining the approbation of his medical colleagues. Feingold would continue to speak to medical audiences, such as the AAP in 1977, and publish his ideas in medical journals, but these tended to be less renowned journals such as

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Ecology of Disease and the *Delaware Medical Journal*.⁴⁶⁹ Feingold's post-1975 publications in the *Journal of the American Society for Preventive Dentistry*, the *American Journal of Nursing*, the *Journal of Learning Disability*, the *International Journal of Dermatology*, *Academic Therapy* and the *International Journal of Offender Therapy and Comparative Criminology* also suggest that dentists, nurses, special educators, dermatologists, psychologists and professionals working in the criminal justice system were among the professionals whom Feingold wished to influence.⁴⁷⁰

Nevertheless, Feingold's efforts after 1974 tended increasingly to focus on parents, the media and other allied health and education professionals who might appreciate his theory. Feingold gave dozens of media interviews, presented his views at countless speaking engagements and addressed the public on the radio and television as well.⁴⁷¹ Moreover, he was not selective when it came to interviewing with less reputable publications, such as the *National Inquirer* or *Reader's Digest*, as Jane Hersey described:

He was in Washington and he appeared on some ... TV show, and we were there in the audience and after the show ... the studio ... had this big beautiful black limousine sitting outside waiting to take him wherever he was going. And there was also there a reporter from *Reader's Digest* - sad to say *Reader's Digest* never published whatever it was the reporter wrote - but the reporter there really wanted to speak to Dr. Feingold, and there wasn't much time, much opportunity, and the reporter said, somewhat embarrassed, 'If you let me drive you back, you know we could have sometime together', he said, 'I have this little Volkswagen bug, you know', [laughs] and Feingold, I mean it wasn't even an issue, you know, he didn't stop and think for a minute, okay do I want to ride in this little jalopy or do I want to go in this fancy limo? ... It was a non-issue and he didn't even let the guy finish, they just started walking to the Volkswagen bug.⁴⁷²

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⁴⁷⁰ Ben F. Feingold, 'Food Additives in Clinical Medicine', *International Journal of Dermatology* 14 (1975), 112-4; Ben F. Feingold, 'Hyperkinesis and Learning Disabilities Linked to Artificial Food Flavors and Colors', *American Journal of Nursing* 75 (1975), 797-803; Ben F. Feingold, 'Food Additives in Dentistry', *The Journal of the American Society for Preventive Dentistry* 7 (1977), 13-5; Feingold, 'A Critique';

⁴⁷¹ In 1978 Feingold described to a colleague that he had 'actually been in a tailspin with so much to do and so many requests [to lecture about his theory]. Letter from Ben. F. Feingold to Beatrice Trum Hunter, 7 April 1978, Beatrice Trum Hunter Collection, Box 47.

⁴⁷² Hersey was right to lament the fact that *Reader's Digest* never published the story. *Reader's Digest* was the world's best-selling magazine during the period and remains so today, reaching millions of households in the United States alone. Jane Hersey, Telephone Interview.

Despite his eagerness with the media, Feingold's shift towards a popular audience also reflected his belief that he had been slighted by his colleagues and the *AMA* and other medical associations. According to most sources Feingold was surprised and disappointed by the decisions of *JAMA*, the *Western Journal of Medicine* and the *BMJ* not to publish his findings on hyperactivity and food additives. Alex Schauss, commenting on how Feingold reacted to such prestigious journals rejecting his submissions, stated that Feingold:

was dismayed. Dejected. I remember how he looked at his wife as he tried to rationalize it. You could see the tears in her eyes. At times he would just stare at the San Francisco harbor from his high rise apartment when looking back at his career and realizing that it had no influence on his peers ... He became disillusioned with these journals afterwards. He realized that politics played a more important role than science. He said, 'If I had a drug for these kids, I would have no problem having these same journals accept my papers'.⁴⁷³

Other associates of Feingold's have also stressed that he was 'shocked by the rejection and criticism', responses which 'left him bewildered' and 'pained'.⁴⁷⁴

Because of such disillusionment, Feingold was not reluctant to recount his humiliating experiences of trying to publish his theory in prestigious medical journals to various audiences, and, when doing so, tended to express a degree of bitterness with regards to the episode. In a response to a 1977 criticism of his theory by Robert L. Sieben, a Connecticut-area paediatric neurologist who accused Feingold of not submitting his idea to the scrutiny of his peers, Feingold first defended his reputation by stating that 'as a practitioner of international reputation for over fifty years, I am well acquainted with the proprieties of medical practice, so that moralistic and ethical innuendos are completely unjustified'.⁴⁷⁵ He then proceeded to outline why he chose to write for the broader public, stating that 'it may be

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of special interest to Dr. Sieben to learn that in each instance, 1973, 1974, and 1975, organized medicine rejected my manuscripts for publication' and that *Why Your Child is Hyperactive* 'was written for a general public following the rejection of my manuscripts'.⁴⁷⁶

Feingold expressed a similar degree of resentment in response to a March 1975 syndicated newspaper column by Frederick Stare (1910-2002), a prominent nutrition scientist and the founder of the Department of Nutrition at Harvard University:

Dr. Stare resorts to additional innuendos which have been copied repeatedly by the lay press and at times even scientific publications. Dr. Stare states: 'Interestingly, to our own knowledge Dr. Feingold has not published a single paper in the medical literature so physicians and scientists can evaluate his results.' The word 'interestingly' seems meant in this context to imply I have purposely withheld publication, an implication that is strengthened by his next statement, 'He apparently preferstalk shows.' Dr. Stare fails to report that my initial presentation was by invitation from organized medicine – the AMA and the California Medical Association – that both the AMA and the California Medical Association rejected for publication the manuscripts of my presentations in 1973, 1974 and 1975. It was following the rejection in 1974 that I accepted an invitation from the publisher [Random House] to author a book *Why Your Child Is Hyperactive* for the general public. Dr. Stare and the other critics fail to mention that since 1973 I have authored twelve publications on the subject of food additives in various scientific periodicals. As for my appearances on talk shows, I must point out to Dr. Stare and his cohorts that practically all the publicity which has led to worldwide awareness of this new modality for hyperkinesis was initially generated by the press conferences scheduled by the AMA and California Medical Association. Furthermore, I continue to feel privileged whenever the media extends an invitation to me to explain my research directly to the public.⁴⁷⁷

Feingold's sharp responses to Sieben and Stare highlight how he resented being seen as a publicity seeker, and not an experienced, honest and responsible physician. By indicating the AMA's role in publicising his theory, as well as the fact that he accepted Random House's invitation to publish his hypothesis only after he had been thrice rejected for publication in leading medical journals, Feingold emphasised that it was not his decision to take his ideas directly to the public without first gaining the approval of the medical community. Moreover,

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the scientific content in *Why Your Child is Hyperactive*, Feingold's unwillingness to link his research with that of food allergists and clinical ecologists, as well as his ongoing efforts to respond to his medical critics and publish his ideas in less renowned journals, suggest that, while he was discouraged by the response of the medical community, he still wanted to be considered as a respected scientist. Although Feingold increasingly shifted his efforts to convincing the public, he nevertheless hoped and even anticipated that his theory would eventually be accepted by his medical peers. As he wrote in a letter to a colleague in late 1979: 'We are making progress, but slower than I would like. The professional climate is gradually changing, and they are beginning to grasp my basic hypothesis.'⁴⁷⁸

Despite Feingold's desire to be taken seriously by his colleagues, there were other, more pragmatic, reasons for him to disseminate his theory through the popular media. One of the demands of scientists who learned of Feingold's hypothesis, both those who were optimistic and those who were sceptical, was that he demonstrate the efficacy of his diet through double-blind clinical trials.⁴⁷⁹ Such trials, for example the ones conducted by Leon Eisenberg and C. Keith Connors during the 1960s to test the effect of Ritalin on hyperactive children, were intended to determine both the efficacy and the safety of proposed treatments.⁴⁸⁰

Feingold, however, had not conducted such trials. In criticising Feingold's decision to report his theory before conducting such trials, prominent New Zealand paediatrician John Werry pointedly stated:

I personally feel there is no greater breach of medical ethics than that of foisting a potentially worthless or dangerous treatment on to a credulous public. Theirs may be the right to believe in magic and panaceas but ours as a profession is to

⁴⁷⁸ Ben F. Feingold, Letter to Beatrice Trum Hunter, 26 December 1979, Beatrice Trum Hunter Collection, Box 47; Anonymous, In-Person interview, 8 December 2007.

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act responsibly, cautiously and scientifically, though not prejudicially. Why should all of my U.S. colleagues in paediatric psychopharmacology research, no more than a handful, have to drop their work to show a clamouring public that Feingold's hypothesis is or is not correct and is or is not safe. Surely the obligation is his before he announces it to the public?⁴⁸¹

Although Feingold had expressed some interest in subjecting his theory to controlled clinical trials when he initially reported it in 1973, his interest and faith in this process waned soon after. His decision to write *Why Your Child is Hyperactive* before the publication of any such trials, conducted by himself or others, is indicative of this. One of the reasons for this was Feingold's impatience to get his ideas out to the public, impatience due largely to his advanced age and health concerns. Not only was Feingold in his mid seventies, he had already undergone two surgeries for cancer and experienced ongoing heart problems that would see him have a pacemaker operation in 1979.⁴⁸² He also commented to a journalist that it would take as many as thirty to fifty years to prove unequivocally through trials that his hypothesis was correct.⁴⁸³ Some of Feingold's colleagues, such as Bernard Weiss, nevertheless urged Feingold to conduct controlled clinical trials, but Feingold demurred, citing his age.⁴⁸⁴ Moreover, according to Conners:

Dr. Feingold at 75 is a man in a hurry. He once told me while we were on a radio program together, "I don't have time for sacred cows of science, the double-blind placebo controlled trials" ... Rather than support these assertions with laborious and time-consuming studies, he preferred to take his message directly to the consumer.⁴⁸⁵

Given the fact that Feingold attempted to present his ideas to his medical colleagues, Conners' assertion that Feingold 'preferred to take his message directly to the consumer' is not altogether accurate. Regardless, there was another, more epistemological reason that

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⁴⁸² Letter from Ben F. Feingold to Beatrice Trum Hunter, 26 December 1979, from the Beatrice Trum Hunter Collection, Box 47; Friedman, *History of the Kaiser Permanente Medical Care Program*.

⁴⁸³ Tom Monte, 'Feingold Diet: A Precious Commodity to Hyperactive Children?' *Chicago Tribune*, 5 January 1980, W11-W12, at p. W12.

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⁴⁸⁵ Feingold would have actually been in his early eighties when Conners wrote this statement in 1980.

Feingold questioned the desirability of conducting controlled clinical trials. Specifically, Feingold's experience trying to develop an allergen to desensitise people allergic to flea bites had demonstrated to him many of the pitfalls and frustrations of trying to derive knowledge through scientific experimentation. In one article, for example, Feingold elaborated on the difficulty of even determining if in fact his flea bite allergy patients were suffering from flea bites:

One unfortunate factor in most of this work has been the dependence of workers on subjective reports from patients regarding the insect causing clinical symptoms. In our experience many patients never see the insect biting them except in the case of mosquitos and biting flies. Inconspicuous insects, such as fleas and bedbugs, may possibly bite these people for years without being noticed, and even when seen may be misidentified. During our work individuals have brought "fleas" to the clinic which have proven to be anything from sawtooth grain beetles to small weed seeds. Needless to say, under these conditions it is difficult to ascertain whether clinical lesions are due to actual flea bites, and, if hyposensitization is attempted, whether any reported relief is due to treatment or to the cessation of flea activity.⁴⁸⁶

Feingold proceeded to explain that no fleas were to be found in many of the houses of patients who complained of flea bite allergies. Moreover, Feingold's flea bite research, while important to the development of allergy theory, failed to develop techniques for desensitising people to flea bite allergies.⁴⁸⁷ Not only was it difficult to design such experiments and eliminate potentials for error, it was also clear to Feingold that such experiments did not always yield the results that were intended.

As an allergist, Feingold was also aware of the potential for individual differences in patients, differences that could affect the outcome of even carefully designed and controlled trials. His research during the 1960s on psychosomatic allergy, for example, suggested to him that 'psychological factors are of importance in understanding allergic illnesses', but that it was

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‘unlikely that any typical allergic pattern will emerge’ and that the allergy population was ‘quite heterogeneous’.⁴⁸⁸ In other words, not only was it complicated to determine the degree to which allergic symptoms were dependent on psychological factors, but it was also difficult to develop a schema of reference from which to assess such influences. Environmental differences could also play a role, as described by Alice Friedman, a paediatric allergist who worked for Feingold during the late 1960s and early 1970s:

It’s extremely difficult in allergy and allergy treatments to have a firm scientific basis because what individuals do has such an important bearing. You know, if you smoke and irritate all your membranes very obviously your symptoms are going to be there no matter what else you do. If you have five dogs and three cats and you’re sensitive to dander, you’re going to have problems no matter how you’re treated for your hayfever. Allergy, more than almost anything else, gets into what you do in your everyday life. To document all these things is very difficult. If you live in a moldy house, which can certainly happen in San Francisco, you may have terrible problems that nobody can solve until you get out of that house. So there are so many factors that are parts of everyday living that can affect the outcome of your scientific endeavors. But it’s extremely difficult to document these things.⁴⁸⁹

Given such experiences, it is somewhat understandable that Feingold might find conducting controlled clinical trials a daunting, laborious and unpromising procedure. As will become apparent in Chapter 9, researching the Feingold diet was fraught with such methodological concerns. In addition, however, Feingold had one other reason for refusing to engage in clinical trials by the time he decided to publish *Why Your Child is Hyperactive*. This was a strong belief, reinforced by ever-increasing number of clinical encounters, that his diet worked. Although Feingold’s naturally conservative tendencies were reflected in his initial hesitation to promote his idea before he was certain of its validity, such caution was eventually overcome by Feingold’s high degree of self-assurance and confidence.⁴⁹⁰ In other

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⁴⁹⁰ Feingold’s confident nature has been echoed in descriptions of him by Dov Michaeli and Alice Friedman, two of his employees, although they have interpreted such characteristics in a more negative fashion, calling him ‘despotic’ and ‘exceedingly autocratic’. Friedman, however, also described him as ‘very positive’ in the sense that he had great faith in his own ideas, and had the ability to convey this faith to others. This attribute might partly explain why she also described him as an excellent teacher.

words, once Feingold believed that his theory was correct, he steadfastly defended it and was not likely to change his opinions in the face of results from double-blind controlled trials.

Conclusion

After initial encouragement, the AMA and other medical associations were ultimately unwilling to provide Feingold with a venue to disseminate his theory to his fellow physicians. Disillusioned and frustrated by this about face, and doubtful that much would be gained by conducting the trials demanded by physicians such as Werry and Sieben, Feingold became more willing to disseminate his theory directly to the public, often using the media to do so. After half a century of being a conservative and respected leader in the paediatric allergy community, Feingold chose to become a radical figure, a pariah who eschewed the ‘sacred cows of science, the double-blind placebo controlled trials’ and took his message directly to parents. Although he would continue to couch his theory in scientific language and associate it with those of others studying neurology and toxicology, Feingold realised that, if his theory was ever to achieve medical respectability, it would be because of bottom-up pressure from parents, journalists, activists, and unorthodox physicians, rather than top-down recommendations from bodies such as the AMA.

According to Roger Cooter and Stephen Pumfrey, the popularisation of scientific theories can transform how such ideas are understood, not only by the public, but also by the scientific community. Scientists often enrol a ‘network of alliances’ in order to communicate their theories to popular audiences and, during this process of translation, the meaning and application of their ideas may change.⁴⁹¹ As Cooter and Pumfrey explain, ‘there is no reason to suppose that popular science takes the form intended by its popularizers’, largely ‘because it is developed by its recipients for different purposes’.⁴⁹² Depending on what these purposes might be, and according ‘to its position and influence in the “network”, the public alters the kind of science pursued in future’.⁴⁹³

⁴⁹¹ Roger Cooter and Stephen Pumfrey, ‘Separate Spheres and Public Places: Reflections on the History of Science Popularization and Science in Popular Culture’, *History of Science* 32 (1994), 237-67, at p. 250.

⁴⁹² *Ibid.*, 249-50.

⁴⁹³ *Ibid.*, 250.

In the seemingly parallel case of Hans Selye's theories about the physiological impact of stress, popularisation had a considerable effect on how stress would be conceived by both the public and scientists. Russell Viner has employed Bruno Latour's actor network theory to explain how Hans Selye attempted to promote his ideas about stress and the general adaptation syndrome, 'a universal truth regarding the relationships of organisms with their environment, a truth he would sell to whoever would listen'.⁴⁹⁴ Selye found that, despite initial interest during the 1930s and 1940s, his colleagues in laboratory research felt that his theory was 'too vague and teleological to be scientifically credible', and questioned his research methods and personality.⁴⁹⁵ Selye then enrolled allies in the popular domain, first in the field of military medicine, and later in the conservative American establishment, who were enticed by his notion that societal strife could manifest itself in disease.⁴⁹⁶ Although the general idea that stress played a key role in affecting physiology would become accepted by numerous scientific disciplines during the 1970s, many specifics underlying Selye's theory were undermined and stress became a more elastic concept than Selye had originally envisioned.⁴⁹⁷

In the case of the Feingold diet, popularisation had more of an impact on the reception of Feingold's hypothesis than its actual substance. This is partly because, as a clinician, the interests Feingold had in disseminating his hypothesis were roughly parallel to those of the parents who employed his diet; both were dissatisfied with contemporary treatments of hyperactivity and desired an alternative. Feingold and parents equally desired a dietary regimen that was comprehensive, but not unnecessarily restrictive. Feingold's hypothesis was also based more on the accumulation of clinical experience, which was itself dependent

⁴⁹⁴ Russell Viner, 'Putting Stress in Life: Hans Selye and the Making of Stress Theory', *Social Studies of Science* 29 (1999), 391-410, at p. 394.

⁴⁹⁵ *Ibid.*, 396.

⁴⁹⁶ *Ibid.*, 399-402.

⁴⁹⁷ *Ibid.*, 402-5.

upon the ability of patients to observe and recall how and when their symptoms arose, than on an underlying theoretical model. As such, Feingold was happy to work with parents and with FAUS to refine the diet, for example, adding preservatives to the diet after *Why Your Child is Hyperactive* was published, but resisting calls to include white sugar to the list of banned substances. After Feingold's death, FAUS continued to adjust the list of approved substances, often in response to parent suggestions. Nonetheless, FAUS adhered closely to Feingold's general principle that the diet had to be kept as liberal as possible, and stubbornly rejected suggestions to ban food products criticised by other groups, such as clinical ecologists. Although Feingold's theory was shaped by parents, his most important allies, it is probable that this also would have been true had he not decided to write a popular book.

The popularisation of the Feingold diet, however, did expose the belief of many physicians during the 1970s, such as Seiben, Stare and Werry, that untested medical ideas posed a danger to the public. In some ways, such thinking could be seen as protecting patients from quackery and snake oil salesmen, not to mention unscrupulous drug companies. But it also implied that patients and their families should not be involved in the assessment or formulation of novel medical ideas, and that members of the public were not capable of informing medical opinion about the provision of treatment. Not only was such thinking relatively naïve, in that it underestimated the agency patients and their families exhibited in selecting or refining medical advice, it also failed to recognise that when patients were dissatisfied with a particular medical approach they would simply look elsewhere. As the following chapter demonstrates, dissatisfaction with conventional explanations for and treatments of hyperactivity led patients and their families to consider alternative approaches.

Chapter 6

Debate, Division and Dissatisfaction: Medical and Popular Responses to Hyperactivity

Without the support of the AMA, it was difficult for Feingold not only to gain the credibility he desired for his diet, but also to communicate his idea to parents via their physicians. Fortunately for Feingold, Random House's offer to publish his theory in 1974 provided him with an ideal opportunity to present his idea directly to parents and foment a debate about the aetiology and treatment of hyperactivity. Random House's interest, however, also affirmed that while the mainstream medical community was unwilling to endorse Feingold's hypothesis, the public was intrigued by the notion that food additives could cause hyperactivity. As the following two chapters suggest, such interest was twofold, in that it reflected not only the public's concern about increasing rates of hyperactivity, and the controversial ways in which the disorder was treated, but also growing alarm about chemicals in the food supply.

Although these issues were quite separate on the surface, they both reflected a growing distrust of corporate America, represented in these cases by the pharmaceutical and food manufacturing industries, as well as the professional associations and government departments, such as the AMA and the FDA, that were thought to safeguard the public against corporate irresponsibility. As Canadian psychiatrists Ivan Williams and Douglas Cram suggested, commenting on how concern about contemporary explanations and treatment of hyperactivity contributed to the popularity of the Feingold diet, the 'radical critique fits into the spirit of the times when American society came under systematic questioning and attack'.⁴⁹⁸ At one level, the Feingold diet provided a particular treatment for

⁴⁹⁸ Williams and Cram also postulated that the popularity of the Feingold diet was due to the following factors: 1) it was an alternative to drugs; 2) it shifted guilt from parents to food industry; 3) it removed blame from

a specific childhood disorder, but at another level, it also represented a critique of American psychiatry and of corporate practices regarding the environment and the food supply. Interest in the Feingold diet was a grassroots response to a medical problem at a time, during the early 1970s, when Americans were increasingly disillusioned with their political leadership in the light of crises such as the Vietnam War, the Watergate scandal and energy shortages.

Hyperactivity emerged during a time of considerable cultural, educational, demographic and political turbulence within American society, but also during a time when the American psychiatric community was undergoing upheaval and was fraught with interdisciplinary strife. The psychiatric community of the 1960s and 1970s consisted largely of three primary disciplines: psychoanalysis; social psychiatry; and biological psychiatry. The theoretical underpinnings and modes of treatment could overlap in practice: for example, psychoanalyst George A. Rogers found that Ritalin facilitated psychotherapy in his neurotic patients.⁴⁹⁹ However, each approach essentially represented a significantly different way of understanding mental illness, and psychiatrists typically favoured one methodology over the others. The profound differences inherent in each discipline's approach to mental illness were, in turn, reflected in their explanations of what caused hyperactivity. Although some psychiatrists spoke of hyperactivity as being multi-causal, and suggested that a variety of treatments be used to help hyperactive children, most research papers stressed the validity of one approach as opposed to others and, in clinical practice, psychiatrists tended to privilege one treatment modality over another.

schools; 4) it fitted the growing consciousness about ecology and pure foods; and 5) enabled parents to be primary therapeutic agent, as they control the diet. J. Ivan Williams and Douglas M. Cram, 'Diet in the Management of Hyperkinesis: A Review of the Tests of Feingold's Hypotheses', *Canadian Psychiatric Association Journal* 23 (1978), 241-8, at p. 242.

⁴⁹⁹ George A. Rogers, 'Methylphenidate Interviews in Psychotherapy', *American Journal of Psychiatry* 117 (1960), 549-50.

Although by the early 1970s most psychiatrists believed that hyperactivity was a neurological dysfunction passed on genetically or caused by brain damage, they were unable to provide specifics on the mechanisms of the dysfunction, and psychoanalytic, social and environmental explanations were common.⁵⁰⁰ Whereas psychoanalysts blamed strained family relationships for hyperactivity, social psychiatrists looked to social conditions such as poverty, over-crowding and crime-infested neighbourhoods.⁵⁰¹ Feingold's hypothesis differed considerably from these theories, but what it had in common with them, as well as with other ecologically-based theories, was the notion that some aspect of American society, whether it be rooted in family structure, socio-economic conditions, technological change or exposure to chemicals, was pathological to children.

Given the unwillingness of American psychiatrists to compromise on the cause of hyperactivity, it is interesting that Feingold himself acknowledged that hyperactivity had a social and interpersonal dimension. It is possible that, had American psychiatry truly agreed that hyperactivity was a multi-dimensional phenomenon, the Feingold diet would have been much less controversial and, instead, viewed as yet another facet of what was a complicated and recalcitrant disorder. Feingold's acceptance of certain elements of psychoanalytic, social and biological psychiatry made his theory appear more acceptable to parents who were less dogmatic about psychiatric theory than American psychiatrists.

Debates about the incidence of hyperactivity, as well as what the disorder actually represented (abnormal behaviour versus an example of coercive social control), might not

⁵⁰⁰ R. M. Gibson, 'Hyperkinesis - Revisited', *University of Michigan Medical Center Journal* 34 (1968), 213; F. A. Mettler, 'Morphologic Correlates of Azide-Induced Hyperkinesis and Hypokinesis', *Transactions of the American Neurological Society* 93 (1968), 141-4; Oliver David, Julian Clark, and Kytja Voeller, 'Lead and Hyperactivity', *Lancet* 300 (1972), 900-3; M. W. Sauerhoff and I. A. Michaelson, 'Hyperactivity and Brain Catecholamines in Lead-Exposed Developing Rats', *Science* 182 (1973), 1022-4; Oliver J. David, 'Association between Lower Level Lead Concentrations and Hyperactivity in Children', *Environmental Health Perspectives* 7 (1974), 17-25; E. K. Silbergeld and A. M. Goldberg, 'Hyperactivity: A Lead-Induced Behavior Disorder', *Environmental Health Perspectives* 7 (1974), 227-32;

⁵⁰¹ , 541-59.

have mattered a great deal to the parents of hyperactive children. By seeking a solution to their child's behaviour, parents implicitly acknowledged that something was amiss with their child. Nevertheless, these disputes, as well as the wide range of opinion represented, highlighted the controversial nature of hyperactivity and suggested that its diagnosis was not straightforward. Parents were also concerned that the methods psychiatrists used to treat hyperactivity were ineffective, inappropriate or dangerous. By the 1970s the most common method for treating hyperactivity was with stimulant drugs, such as Ritalin, Cylert and Dexedrine, although psychotherapy, family counselling and behavioural therapy were also treatment alternatives. Despite the variety of explanations and treatment methods for hyperactivity, many parents were hesitant to accept conventional explanations or solutions for their child's behavioural problems and found that Feingold's proposition provided a much more straightforward explanation.

This chapter begins by exploring the debates which shaped American understandings of hyperactivity. It investigates how psychoanalysts, social psychiatrists and biological psychiatrists described and treated hyperactivity in fundamentally different ways and were reluctant to develop a pluralistic understanding of the disorder that combined elements of psychoanalysis, social theory and neurology. Biological psychiatry won the debates about how to conceptualise hyperactivity not so much because its approach was more scientifically valid or accurate, but rather because its methods were less expensive, time consuming and complicated than those of its rivals. The chapter concludes by demonstrating how, although biological theories of hyperactivity were to dominate psychiatric understandings of the disorder by the 1970s, large sections of the American public were unimpressed with such explanations and were uncomfortable with using drugs to treat their children's behavioural

problems. Dissatisfaction with the primary methods of treating hyperactivity led parents to consider alternative approaches, and, during the 1970s, the Feingold diet was by far the most successful of these alternatives.⁵⁰²

Psychoanalysis: The ‘most productive and cohesive theory available’

Following the Second World War, psychoanalysis was the dominant discipline within American psychiatry, influencing not only clinical practice and research, but also how the public perceived psychiatry.⁵⁰³ For example, the editorial board of *JAACP*, founded in 1962 to reflect the increasing interest in child psychiatry, consisted primarily of psychoanalysts, and most of the articles published by the journal during the 1960s were oriented towards psychoanalysis.⁵⁰⁴ In a special series on childhood behavioural problems in the second volume of the journal, all articles were based in psychoanalytic theory, including those by Eveleen N. Rexford, the series’ editor.⁵⁰⁵ Psychoanalytic explanations for childhood disorders dominated the 1968 publication of *DSM-II*, including the description of the

⁵⁰² Although there were other theories during the 1970s which linked chemical exposure to hyperactivity, these failed to become as popular or controversial as Feingold’s hypothesis. Despite attracting even less attention since the 1970s, the debate regarding lead and hyperactivity has recently re-emerged. The most recent researchers to study the link have emphasised the importance of identifying the genotype which indicates sensitivity to low-level lead exposure. Similarly, Feingold believed that certain children were genetically predisposed to be highly sensitive to food additives. Joel T. Nigg, G. Knottnerus, M. Martel, K. Cavanagh, W. Karmaus and M. Rappley, ‘Low Blood Levels Associated with Clinically Diagnosed Attention-Deficit/Hyperactivity Disorder and Mediated by Weak Cognitive Control’, *Biological Psychiatry* 63 (2008), 325-31.

⁵⁰³ Grob, *From Asylum to Community*, 100; Nathan Hale, *The Rise and Crisis of Psychoanalysis in the United States* (New York: Oxford University Press, 1995); Edward Shorter, *A History of Psychiatry* (New York: John Wiley and Sons, 1997); John J. Leveille, ‘Jurisdictional Competition and the Psychoanalytic Dominance of American Psychiatry’, *Journal of Historical Sociology* 15 (2002), 252-80, at p. 252.

⁵⁰⁴ According to Andrew Lakoff, the journal shifted its focus away from dynamic approaches and to biological psychiatry in 1976 when Melvin Lewis replaced Rexford as editor. The first article of the new era was Dennis P. Cantwell’s ‘Genetic Factors in the Hyperkinetic Syndrome’. Lakoff, ‘Adaptive Will’, 155.

⁵⁰⁵ For example, Eveleen N. Rexford, ‘A Developmental Concept of the Problem of Acting Out’, *Journal of the American Academy of Child Psychiatry* 2 (1963), 6-21; Charles A. Malone, ‘Some Observations on Children of Disorganized Families and Problems of Acting Out’, *Journal of the American Academy of Child Psychiatry* 2 (1963), 22-49, at pp.22-3; David E. Reiser, ‘Observations of Delinquent Behavior in Very Young Children’, *Journal of the American Academy of Child Psychiatry* 2 (1963), 50-71.

hyperkinetic reaction of childhood.⁵⁰⁶ Psychoanalysts also regularly took up the presidency of the APA during the post-war period.⁵⁰⁷

Psychoanalysts tended to guard jealously their hegemony over American psychiatry, especially as rival psychiatric theories became popular during the 1950s and 1960s. A letter written by Iowa child psychiatrist Mark Stewart to the editor of *AJP* in 1960 echoed psychoanalytic dominance of American psychiatry, but also implied that not all psychiatrists were happy with the situation. Stewart argued that jobs advertised in the APA's 'Mail Pouch' nearly always stressed the importance of a dynamic orientation, and complained that 'this phenomenon, which unhappily is symptomatic of the general situation of psychiatry today, can make our profession seem ridiculous to other physicians and to scientists in general'.⁵⁰⁸ Although Stewart's criticism foreshadowed the ultimate demise of American psychoanalysis, for many psychiatrists working during the emergence of hyperactivity there was no 'magical belief in some kind of correspondence between psychical processes and central nervous processes'.⁵⁰⁹

Psychoanalysts believed that hyperactivity, like many other psychiatric conditions, was rooted in family dynamics and involved disruption of the superego which, in turn, resulted in poor impulse control.⁵¹⁰ Although this explanation appeared simple superficially, the key for psychoanalysts was to determine what initially caused such disruption in order to provide

⁵⁰⁶ Richard L. Jenkins, 'Classification of Behavior Problems of Children', *American Journal of Psychiatry* 125 (1969), 1032-9, at pp. 1032-3.

⁵⁰⁷ Leveille, 'Jurisdictional Competition', 252-3.

⁵⁰⁸ Mark A. Stewart, 'Correspondence: Dynamic Orientation', *American Journal of Psychiatry* 117 (1960), 85.

⁵⁰⁹ This quotation came from a Swedish-American psychiatrist who lamented the fact that Swedish psychiatrists were turning away from psychoanalysis in favour of biological approaches. His letter to the editor received a curt reply from Olof Kinberg, a Swedish psychiatrist who stated that if Swedish psychiatrists were turning away from psychoanalysis, then they should be complimented for doing so. Olof Kinberg, 'Reply to the Foregoing', *American Journal of Psychiatry* 116 (1959), 84; L. Borje Lofgren, 'A Comment on "Swedish Psychiatry"', *American Journal of Psychiatry* 116 (1959), 83-4.

⁵¹⁰ David E. Reiser, 'Observations of Delinquent Behavior', 50, 53, 67; Rexford, 'A Developmental Concept', 9-10.

effective psychotherapy. As a result, most of the psychoanalytic articles in psychiatric journals during the 1960s about hyperactivity were written in the form of case studies featuring the clinical observations of a single patient. The patient would be introduced along with a detailed description of his or her behaviours, personality, history and family situation. The authors would then describe how they were able to unravel the reasons for the patient's hyperactivity and recount the course of treatment. One instance of this is found in a 1960 edition of the *Archives of General Psychiatry*, in which the story of 'Jean' was described. Jean was a 12-year-old girl whose impulsive behaviour, her psychiatrist determined, was the result of penis envy stemming from the relationship that she had with her father. Jean's impulsivity ceased only when she was able to come to terms with this explanation.⁵¹¹ The root causes of hyperactivity in other children could also originate in the child's weaning, toilet training, adjustment to a new sibling or a response to other types of trauma.⁵¹² In other cases, inappropriate, unhealthy or inadequate relationships with parents were believed to be the problem.⁵¹³

In many ways, case studies were an attractive means by which to depict hyperactivity and the course of psychoanalytic treatment. The reader was provided with a mini-narrative which usually resulted in a happy ending; the child who was so disruptive at the beginning of the case study was usually thriving at both school and home by the end of it. Although sceptics could question the reliability of such descriptions, case studies had an emotional impact upon readers which the impersonal accounts of double-blind clinical trials lacked. As such, and in

⁵¹¹ J. Weinreb and R. M. Counts, 'Impulsivity in Adolescents and its Therapeutic Management', *Archives of General Psychiatry* 2 (1960), 548-58, at pp. 549-50.

⁵¹² Rexford, 'A Developmental Concept', 10-1; Reiser, 'Observations of Delinquent Behavior', 53; Alexander Thomas, Herbert Birch, Stella Chess and Lillian C. Robbins, 'Individuality in Responses of Children to Similar Environmental Situations', *American Journal of Psychiatry* 116 (1960), 798-803, at p. 798.

⁵¹³ Rexford, 'A Developmental Concept', 11; Adelaide M. Johnson and S.A. Szurek, 'The Genesis of Antisocial Acting Out in Children and Adults' in I. N. Berlin and S. A. Szurek (eds.), *Learning and its Disorder: Clinical Approaches to Problems of Childhood* (Palo Alto, California: Science and Behavior Books, 1965), 120-38, at p. 136; Esther S. Battle and Beth Lacey, 'A Context for Hyperactivity in Children over Time', *Child Development* 43 (1972), 757-773, at pp. 757, 772.

keeping with a similar tradition in allergy, Feingold also used case studies to great effect in *Why Your Child is Hyperactive*.

His use of case studies notwithstanding, Feingold, like many parents and other physicians, was less partial to how psychoanalysts explained hyperactivity. Although Feingold agreed that the stress and disruption of having a hyperactive child could strain relationships both at home and at school, even compromising the efficacy of the Feingold diet, such problems were at root caused by food additives.⁵¹⁴ For parents of hyperactive children, including many of those who would employ the Feingold diet, psychoanalytic explanations of hyperactivity were often confusing and contradictory, and seemed to imply, if not explicitly state, that parents were to blame for their child's hyperactivity.⁵¹⁵ Mothers, in particular, were singled out as being responsible for causing behaviour problems in their children. According to journalists Barbara Ehrenreich and Deirdre English, 'by the mid-twentieth century the experts were grimly acknowledging that despite constant vigilance the American mother was failing at her job'.⁵¹⁶ The ubiquity of such notions during the post-war period meant that other physicians would often pin a range of health problems on poor mothering.⁵¹⁷ As Shula Edelkind, a volunteer with FAUS, described when she tried to find help for her son's alarming twitching and tics:

I took him to an ENT doctor who examined him and said, 'There's nothing wrong, Mother, he needs more attention.' I took him to an eye doctor because one of the early tics was looking cross-eyed and looking up and down and around the edges of things and he said, 'There's nothing wrong with his eyes,

⁵¹⁴ Feingold, *Why Your Child is Hyperactive*, 38.

⁵¹⁵ Shula Edelkind, Telephone Interview, 28 January 2008.

⁵¹⁶ Barbara Ehrenreich and Deirdre English, *For Her Own Good: 150 Years of the Experts' Advice to Women* (Garden City, New York: Anchor Books, 1979), 217.

⁵¹⁷ For example, overprotective, smothering mothers were believed to be pathological in the cases of schizophrenia and asthma. While German psychoanalyst Freida Fromm-Reichmann (1889-1957) described the 'schizophrenogenic mother' in 1948, the 'asthmogenic home' was a concept employed by pioneering English allergist John Freeman during the same period. Mark Jackson, "'Allergy Con Amore": Psychosomatic Medicine and the "Asthmogenic Home" in the Mid-Twentieth Century', in Mark Jackson (ed.), *Health and the Modern Home* (New York: Routledge, 2007), 153-74, at pp. 159-65.

Mother, he needs more attention.’ This kid gets all the attention in the family. How could he possibly need more attention?⁵¹⁸

Although Edelkind found that Feingold’s theory could also be interpreted as having an element of mother-blame, in that mothers were largely responsible for feeding their children harmful additives, Feingold and FAUS almost always refrained from criticising mothers and, instead, criticised corporations, medical associations and federal agencies.

Other psychiatric theories, particularly biological psychiatry, not only absolved mothers from blame, but also provided exciting new remedies for psychiatric problems. Drugs such as the anti-psychotic Thorazine and the anti-depressant Miltown were advertised as being able to treat patients ranging from the schizophrenic in the asylum, potentially allowing such a patient to return to the community, to the depressed housewife. As pharmaceutical companies began experiencing success selling such drugs during the late 1950s and 1960s, psychoanalysis was increasingly seen as anachronistic and unscientific. This was particularly pertinent to psychiatrists who desired the same kind of authority and respect that was accorded to other physicians.⁵¹⁹ As child psychiatrist John S. Werry described, encouraging his colleagues to employ ‘pediatric psychopharmacology’, ‘child psychiatry ... is not simply a humanitarian exercise, but an applied biological science’.⁵²⁰

Despite the excitement and sales generated by the new medications, however, many psychoanalysts unreservedly insisted that neurology and psychiatry should not mix. As Albert J. Solnit (1919-2002), one of the first American psychiatrists to specialise in child psychiatry, asserted, ‘there is considerable doubt that the use of research models

⁵¹⁸ An ‘ENT doctor’ is a otolaryngologist, or ear, nose and throat doctor. Shula Edelkind, Interview.

⁵¹⁹ Henry A. Davidson, ‘The Image of the Psychiatrist’, *American Journal of Psychiatry* 121 (1964), 329-34; Robert H. Felix, ‘The Image of the Psychiatrist: Past, Present, and Future’, *American Journal of Psychiatry* 121 (1964), 318-22, at p. 319.

⁵²⁰ John S. Werry, ‘The Use of Psychotropic Drugs in Children’, *Journal of the American Academy of Child Psychiatry* 16 (1977), 446-68, at p. 463.

derived from the physical sciences can be of more than limited usefulness in child psychiatry research'. According to Solnit psychoanalysis was the 'most cohesive and productive theory available'.⁵²¹ Solnit's assertion, however, was increasingly unpopular amongst American psychiatrists, and his article generated a heated response from Leon Eisenberg, who had conducted the first large-scale Ritalin trials with C. Keith Conners. Eisenberg stated that psychoanalysis had 'a constricting influence' on psychiatry and that the psychoanalytic case studies published in journals such as *JAACP* should be replaced with 'epidemiological, pharmacological, and psychological studies'.⁵²²

More important, perhaps, was the practicality of treating the vast numbers of hyperactive children, variably estimated at between five and twenty per cent of the childhood population, with psychotherapy.⁵²³ Psychoanalytic theory required that each case be treated individually or, in the words of an anonymous individual quoted in *AJP*, 'individual psychotherapy is the only treatment that roots out the trouble. You can't apply this on a mass basis.'⁵²⁴ Many psychiatrists recognised, however, that there were 'more people struggling in the stream of life than we can rescue with our present tactics' of employing psychotherapy, and argued that there were nowhere near enough psychotherapists to treat the 'extraordinary numbers of disturbed children in the country'.⁵²⁵

⁵²¹ Albert J. Solnit, 'Who Deserves Child Psychiatry? A Study in Priorities', *Journal of the American Academy of Child Psychiatry* 5 (1966), 1-16, at p. 3.

⁵²² Leon Eisenberg, 'Discussion of Dr. Solnit's Paper "Who Deserves Child Psychiatry? A Study in Priorities"', *Journal of the American Academy of Child Psychiatry* 5 (1966), 17-23, at pp. 20-1.

⁵²³ Anonymous quoted in Henry A. Davidson 'Comment: The Reversible Superego', *American Journal of Psychiatry* 120 (1963), 192-3, at p. 192. Although practitioners of group therapy would object to such a comment, few practitioners recommended group therapy for hyperactivity. One exception was: S. A. Cermak, F. Stein and C. Abelson, 'Hyperactive Children and an Activity Group Therapy Model', *American Journal of Occupational Therapy* 27 (1973), 311-5.

⁵²⁵ Eisenberg, 'Discussion of Dr. Solnit's Paper', 23; Charles Hersch, 'The Clinician and the Joint Commission Report: A Dialogue', *Journal of the American Academy of Child Psychiatry* 10 (1971), 406-17, at p. 411.

Still others criticised the effectiveness of psychotherapy in treating hyperactivity altogether, charging that ‘unfortunate children with minimal brain dysfunction are still being condemned to months of fruitless and frustrating psychotherapy in various guidance clinics, while guilt and resentment builds upon their bewildered parents’, and that ‘misdirected psychotherapy can be every bit as dangerous as misdirected surgery’.⁵²⁶ While it is important to note that the previous quotation came from an unapologetic supporter of using stimulant therapy to treat hyperactive children, psychoanalysts themselves admitted that psychotherapy was a time-consuming, expensive and emotionally demanding intervention.⁵²⁷ Although some of the families who employed the Feingold diet relied on some form of counselling or psychotherapy to help them deal with their child’s hyperactivity, most typically interpreted such measures as supplemental, often addressing the emotional fallout of coping with a hyperactive child, rather than the behaviour itself.⁵²⁸

Even psychoanalysts who were confident about the efficacy of psychotherapy could find that hyperactive patients were not particularly easy to treat. Psychotherapy required that a patient concentrate, be reflective and follow dutifully the psychotherapist’s suggestions.

Understandably, this was an arduous requirement for hyperactive children to meet.⁵²⁹ One psychoanalyst described how her patient’s ‘hyperactivity increased and all in a manner of a

⁵²⁶ Harold B. Levy, ‘Amphetamines in Hyperkinetic Children’, *JAMA* 216 (1971), 1864-5, at p. 1865.

⁵²⁷ Eveleen N. Rexford ‘Child Psychiatry and Child Analysis in the United States’, *Journal of the American Academy of Child Psychiatry* 1 (1962), 365-84, at p. 381.

⁵²⁸ Bonnie Kowaliuk, Telephone Interview, 5 November 2007; Shula Edelkind, Interview; Colleen Davis, Telephone Interview, 5 February 2008.

⁵²⁹ It is somewhat strange that psychoanalysts rarely suggested play therapy, employed as early as 1926 by child psychoanalyst Anna Freud (1895-1982), as a possible intervention for hyperactive children. Freud saw play therapy as a means to strengthen ego functioning by encouraging children to verbalise what they were feeling at play. Within the bounds of psychoanalytic theory, such therapy would presumably have provided some insights into what drove the impulsivity of the hyperactive child. Anna Freud, *Normality and Pathology in Childhood* (New York: International Universities Press, 1965); Irwin Jay Knopf, *Childhood Psychopathology: A Developmental Approach* (Englewood Cliffs, New Jersey: Prentice-Hall, Inc., 1979), 165-6.

few minutes, she sat on my desk, wrote on the blackboard, and picked her nose excessively'.⁵³⁰ In a market saturated with potential patients, but lacking psychoanalysts, many psychiatrists accused psychoanalysts of turning away hyperactive children because they were difficult to treat successfully.⁵³¹ Psychoanalytic explanations and solutions to hyperactivity might have helped to explain some aspects of the disorder, but as the number of hyperactivity diagnoses expanded rapidly during the 1970s, and as psychiatrists increasingly looked to neurology for the solutions to mental illness, psychoanalysis failed to remain a viable treatment alternative for most families.

Social Psychiatry: 'a preventative psychiatry'

Social psychiatrists not only recognised the impracticality of providing psychoanalysis to hyperactive children, but they also attempted to put forth their own, presumably more pragmatic, solution. During the late 1950s and early 1960s, psychiatrists concerned with preventing mental illness, and concerned with the psychological affect of both geopolitical tension and domestic civil unrest, increasingly looked to society as a source of psychiatric problems. For some social psychiatrists, mental illness could be prevented by alleviating its social causes, particularly poverty, overcrowding, crime, prostitution and substance abuse. For others, social psychiatry was more concerned with providing psychiatric services, often in community mental health centres, to the poor, who were believed to be disproportionately

⁵³⁰ The psychiatrist in question believed that her inability to help this particular patient was an indication that her hyperactivity was neurological, rather than, dynamic, in nature. Paulina F. Kernberg, 'The Problem of Organicity in the Child: Notes on Some Diagnostic Techniques in the Evaluation of Children', *Journal of the American Academy of Child Psychiatry* 8 (1969), 517-41, at p. 537.

⁵³¹ Leon Eisenberg, Anita Gilbert, Leon Cytryn and Peter A. Molling, 'The Effectiveness of Psychotherapy Alone and in Conjunction with Perphenazine or Placebo in the Treatment of Neurotic and Hyperkinetic Children', *American Journal of Psychiatry* 116 (1960), 1088-93, at p. 1092; Sidney Berman, 'Techniques of Treatment of a Form of Juvenile Delinquency, the Antisocial Character Disorder', *Journal of the American Academy of Child Psychiatry* 3 (1964), 24-52, at p. 24; Edmund F. Kal, 'Organic Versus Functional Diagnoses', *The American Journal of Psychiatry* 125 (1969), 1128; Judith Rapoport, Alice Abramson, Duane Alexander and Ira Lott, 'Playroom Observations of Hyperactive Children on Medication', *Journal of the American Academy of Child Psychiatry* 10 (1971), 524-34, at p. 531.

affected by mental illness.⁵³² The corollary to both of these premises was that psychiatrists were expected to be political, as well as medical, actors, and use their authority to encourage social change.

Despite its seemingly radical foundation, the prophylactic strategies espoused by social psychiatrists reflected the beliefs of many psychiatrists during the 1960s, as well as the official policy of the APA, especially with respect to children and adolescents.⁵³³ Many presidents of the APA during the 1960s supported the tenets of social psychiatry and urged their colleagues to study the pathological effects of social problems.⁵³⁴ Much as the founding of *JAACP* was a response to growing interest in child psychiatry, the *International Journal of Social Psychiatry (IJSP)* and *Social Psychiatry* were founded in 1956 and 1966 respectively to reflect such concerns. A more radical social psychiatric journal, entitled *Radical Therapist*, was also founded in 1970.⁵³⁵ The editorial statement which graced the inaugural edition of *Social Psychiatry* not only stressed how such journals would ‘disseminate this growing body of pertinent knowledge’, but also emphasised that the ‘world-wide movement toward a social orientation affects psychiatric practice, education and research’. The

⁵³² The other major initiative of social psychiatry during the 1950s and 1960s was to shift psychiatric services from large asylums to community mental health centres. This allowed the mentally ill, often prescribed new anti-psychotic and anti-depressive medication, to return their own communities, where it was expected that they would be treated more humanely. Although many of the preventative elements of social psychiatry have been forgotten, the positive and negative ramifications of deinstitutionalisation have been the subject of heated debate ever since.

⁵³³ American Psychiatric Association, ‘Position Statement on *Crisis in Child Mental Health: Challenge for the 1970s*, Final Report of the Joint Commission on Mental Health of Children’, *American Journal of Psychiatry* 125 (1969), 1197-1203.

⁵³⁴ C. H. Hardin Branch, ‘Presidential Address: Preparedness for Progress,’ *American Journal of Psychiatry* 120 (1963), 1-11, at p. 10; Ewalt, ‘Presidential Address’, 980; Daniel Blain ‘The Presidential Address; Novaesence,’ *American Journal of Psychiatry* 122 (1965), 1-12, at p. 4; 1-16, at p. 7; Raymond W. Waggoner Sr., ‘The Presidential Address: Cultural Dissonance and Psychiatry’, *American Journal of Psychiatry* 127 (1970), 1-8, at p. 1.

⁵³⁵ *Radical Therapist* was enigmatically renamed *Rough Times* in 1972, possibly to reflect the difficulties social psychiatrists faced in promoting their ideas. The radical wing of social psychiatry nevertheless espoused the same basic premise as other social psychiatrists; mental illness was caused by social factors such as racism, sexism, urbanisation, pollution and poverty. John A. Talbott, ‘Radical Psychiatry: An Examination of the Issues’, *American Journal of Psychiatry* 131 (1974), 121-8, at pp. 121-2.

statement continued to describe the editors' interest in papers which reported 'on the social, cultural and familial determinants of psychic disorders, and their implications for social and psychological treatment'.⁵³⁶

As Sir David Henderson (1884-1965), British psychiatrist and professor of psychiatry at the University of Edinburgh, indicated in his letter supporting the founding of *IJSP*, social psychiatry had its roots in previous generations of psychiatrists, for example, the work of Adolph Meyer during the first decades of the twentieth century in New York.⁵³⁷ Henderson went on to stress that 'social psychiatry is first and foremost a preventative psychiatry. It strives to combat all those causes of social and environmental nature which are manageable' and 'it concerns itself with public welfare in the widest sense'.⁵³⁸ A letter writer in the subsequent issue put matters more bluntly, stating that 'social life is a prolific breeder of mental disease', and that 'we would do, both for the patient and for society as a whole, immediately better if we could go to the roots of these troubles'. Among the pernicious social factors listed by the letter writer were long working hours, poverty, war, racial discrimination, and segregation.⁵³⁹

Taken in the context of the 1960s, in the midst of the civil rights movement, protests against the Vietnam War and the 'New Frontier' and 'Great Society' social policy initiatives of presidents Kennedy and Johnson, it was understandable that many psychiatrists were interested in the preventative concepts of social psychiatry. Indeed, if President Kennedy's 1963 Message to the United States Congress on Mental Illness and Mental Retardation is any indication, social psychiatry seemed poised to challenge psychoanalysis for its hegemony

⁵³⁶ Anonymous, 'Editorial Statement', *Social Psychiatry* 1 (1966), 1.

⁵³⁷ Sir David Henderson quoted in Joshua Bierer, 'Introduction to the Second Volume', *International Journal of Social Psychiatry* 2 (1956), 1-11, at p. 8.

⁵³⁸ Henderson in Bierer, 'Introduction', 8.

⁵³⁹ B. Lieber, 'Letter to the Editor', *International Journal of Social Psychiatry* 2 (1956), 235-7.

during the 1960s. Kennedy's emphasis on eliminating the environmental causes of mental illness, especially poverty, mirrored many of the preventative strategies of social psychiatry, and was a significant aspect of his New Frontier policy.⁵⁴⁰ His stress on the need for psychiatry to rely less on massive, isolated state hospitals, a system he called 'social quarantine', and, instead, shift towards employing more numerous, smaller and localized community mental health centres also echoed the calls of social psychiatrists.⁵⁴¹

By the 1960s social psychiatric research had indicated that preventative social strategies could help to explain and address hyperactivity in children. Researchers found that children brought up in poverty and exposed to vices such as petty crime, prostitution and violence were much more likely to be hyperactive, impulsive and distractible in school and succumb to mental illness later on in life.⁵⁴² Influential child psychiatrists Stella Chess (1914-2007), Alexander Thomas (1914-2003), Sir Michael Rutter (b. 1933) and Herbert G. Birch (1918-1973) also claimed that environmental factors could cause childhood behavioural disorders such as hyperactivity.⁵⁴³ Psychiatrists were discovering that hyperactivity was most commonly diagnosed in poor children, often representing marginalized visible minorities.⁵⁴⁴ Even some biologically-oriented psychiatrists, such as Leon Eisenberg, were sympathetic towards social psychiatric principles. Eisenberg not only lamented that psychiatrists 'neglected prevention in our preoccupation with treatment', but also believed that 'much of

⁵⁴⁰ Kennedy, 'Message from the President', 734-5.

⁵⁴¹ *Ibid.*, 730.

⁵⁴² Grootenboer 'The Relation of Housing', 471; Charles A. Malone, 'Some Observations', 22-3; George E. Gardner, 'Aggression and Violence - the Enemies of Precision Learning in Children', *American Journal of Psychiatry* 128 (1971), 445-50, at p. 446.

⁵⁴³ Stella Chess, Alexander Thomas, Michael Rutter and Herbert G. Birch, 'Interaction of Temperament and Environment in the Production of Behavioral Disturbances in Children', *American Journal of Psychiatry* 120 (1963), 142-8, at p. 147.

⁵⁴⁴ Chess, Thomas and Birch, 'Behavior Problems Revisited', 330; Irving N. Berlin, 'Some Models for Reversing the Myth of Child Treatment in Community Mental Health Centers', *Journal of the American Academy of Child Psychiatry* 14 (1975), 76-94, at p. 84.

the difficult behaviour seen in association with brain damage syndrome stems not from the anatomical deficits, but from the social consequences of personality development'.⁵⁴⁵

Feingold incorporated some aspects of social psychiatry into his theory of hyperactivity.

Social factors might not necessarily cause hyperactivity, but they could undoubtedly exacerbate the disorder and contribute to other behavioural problems. Although 'the ghetto can no longer claim sole ownership ... without question, socioeconomic pressures influence instinctive behavior, and such behavior becomes imprinted on the patterns of the individual ... Deprived infancy and adolescence can add up to a troubled adult.'⁵⁴⁶ What complicated matters for Feingold, however, was that disorders such as hyperactivity were not restricted to the ghetto, but had 'spread to the stamping grounds of the middle class and into wealthy suburbia'.⁵⁴⁷ Socioeconomic inequality might indeed cause much strife, and might even cause mental illness, but it did not entirely explain disorders such as hyperactivity, which were thriving in both lower and middle class populations.

Although the socioeconomic solutions put forward by social psychiatrists garnered a great deal of support during the 1960s, and were reflected in legislation and in research activities, they nevertheless required more political fortitude than psychiatrists could muster, especially after federal funding shifted from the New Frontier and Great Society programmes to waging the war in Vietnam. Indicative of this trend was Brosin's 'Presidential Address' to the APA in 1968/1969. In his 'Response to the Presidential Address' the previous year, Brosin was optimistic about prospects of reducing poverty and improving mental health.⁵⁴⁸ A year later, Brosin's comments were much more cautious. He noted that American involvement in

⁵⁴⁵ Brain damage syndrome was another term used to describe hyperactivity. Eisenberg, 'Discussion of Dr. Solnit's Paper', 23; Leon Eisenberg quoted in Schragar, *et al*, 'The Hyperkinetic Child', 530.

⁵⁴⁶ Ben F. Feingold, *Why Your Child Is Hyperactive* (New York: Random House, [1974] 1996), 160.

⁵⁴⁷ *Ibid*.

⁵⁴⁸ Henry A. Brosin, 'Response to the Presidential Address', *American Journal of Psychiatry* 124 (1967), 7-8.

Vietnam was drawing resources away from mental health programmes and that difficult choices must be made regarding the direction of American psychiatry's focus.⁵⁴⁹ Quoting John W. Gardner, the Secretary of State for Health, Education, and Welfare, Brosin indicated that a 'crunch between expectations and resources' was occurring, especially with regards to 'early childhood education, work with handicapped children, special education for the disadvantaged'.⁵⁵⁰

More important to the parents of hyperactive children, however, was the fact that social psychiatry's focus on prevention did little for children currently experiencing academic and social difficulties due to their behavioural problems. Moreover, as Feingold and many parents were aware, not all hyperactive children came from impoverished backgrounds.⁵⁵¹ Despite Kennedy's endorsement, APA sympathy and the appeal of its preventative philosophy, social psychiatrists had difficulty addressing the escalating rates of disorders such as hyperactivity and, by the 1970s, psychiatrists and parents were looking to more immediate solutions.

Biological Psychiatry: 'no twisted thought without a twisted molecule'

The field of psychiatry which seemed most able to provide the immediate solution to hyperactivity demanded by psychiatrists and parents was biological psychiatry. Drawing on a long tradition of viewing mental illness as a predominantly neurological phenomenon, biological psychiatrists during the 1960s and 1970s were buoyed by recent advancements in pharmacology, particularly the development of impressive psychoactive drugs. By the time the Feingold diet emerged, most, though not all, psychiatrists agreed that there was 'no

⁵⁴⁹ Brosin, 'Presidential Address', 5.

⁵⁵⁰ John W. Gardner quoted in Brosin, 'Presidential Address', 5.

⁵⁵¹ David J. Franks, 'Ethnic and Social Status Characteristics of Children in EMR and LD Classes' *Exceptional Children* 37 (1970-1971), 537-8.

twisted thought without a twisted molecule'.⁵⁵² With regards to hyperactivity, biological psychiatrists looked to the brain and its functioning for the causes of the disorder, and employed not only stimulant drugs, but also tranquilisers and anti-depressants as treatment.⁵⁵³ For many psychiatrists, biological psychiatry's emphasis on the neurobiological causes of mental illness gave the profession renewed respectability within the broader medical community, something that they believed was lacking during the years when psychoanalytic interpretations of mental illness dominated.⁵⁵⁴

Although the shortcomings of psychoanalysis and social psychiatry with regards to treating hyperactivity helped to create a vacuum in which neurological approaches to the disorder could flourish, there were other key factors which contributed to biological psychiatry's dominance of hyperactivity. First, biological psychiatrists could point to a tradition, dating back especially to the epidemic of post-encephalitic disorder during the 1920s, of viewing childhood behavioural problems as neurological phenomena. They could also demonstrate, by highlighting the work of Charles Bradley during the late 1930s, that there was a long history of treating disturbed children with stimulants. The first significant trial of methylphenidate by C. Keith Conners and Leon Eisenberg in 1963, for example, pointed to the work of Bradley and some of his followers.⁵⁵⁵

Similarly, biological psychiatrists had key allies in pharmaceutical companies such as Ciba, the manufacturers of Ritalin, which were understandably interested in taking advantage of, if not over-estimating, the epidemic of hyperactivity. Not only did Ciba fund research and conferences on hyperactivity, they also produced films and pamphlets about the disorder, and

⁵⁵² John I. Langdell, 'Phenylketonuria: Some Effects of Body Chemistry on Learning', *Journal of the American Academy of Child Psychiatry* 6 (1967), 166-73, at p. 166.

⁵⁵³ Rapoport, *et al.*, 'Playroom Observations', 524.

⁵⁵⁴ John S. Werry, 'An Overview of Pediatric Psychopharmacology' *Journal of the American Academy of Child Psychiatry* 21 (1982), 3-9, at p. 3.

⁵⁵⁵ Conners and Eisenberg, 'The Effects of Methylphenidate', 458.

raised awareness of the disorder at Parent Teacher Association (PTA) meetings during the late 1960s and early 1970s.⁵⁵⁶ Such marketing not only helps to explain the rise in rates of hyperactivity, but it also hints at why Ritalin became ‘the treatment of choice [despite] ... very little empirical basis for its supposed superiority’ to other drugs such as dextroamphetamine.⁵⁵⁷ Although the Convention on Psychotropic Substances in 1971 somewhat curtailed the practice of marketing directly to parents and teachers, physicians remained a target of drug companies, as pharmaceutical advertising in medical journals increased enormously during the 1960s and 1970s.⁵⁵⁸ As historian Nancy Tomes has described:

under the American patent system, drug companies had roughly twenty years to profit from a new prescription drug; to build market share in a brutally competitive industry, they had a strong incentive to court physicians aggressively. Doctors were deluged with advertisements through the mail and the pages of their medical journals. ... Whereas once doctors had been a relatively small side specialty in drug advertising, they now became its main target.⁵⁵⁹

Perhaps most crucial to the acceptance of biological interpretations of hyperactivity, however, were two final factors, one relating to how biological psychiatrists accounted for hyperactivity, and the other concerning the treatment of the disorder. First, by treating hyperactivity as a genetic, neurological condition, biological psychiatrists abandoned the tradition, implicit in both psychoanalytic and social psychiatric interpretations of the disorder,

⁵⁵⁶ Don Mahler, ‘Review of the Film: *The Hyperactive Child*’, *Exceptional Children* 38 (1971-1972), 161; Schrag and Divoky, 80-4.

⁵⁵⁷ Dorothea M. Ross and Sheila A. Ross, *Hyperactivity: Research, Theory, and Action* (New York: John Wiley & Sons, 1976), 99.

⁵⁵⁸ Ilina Singh, ‘Bad Boys, Good Mothers, and the Miracle of Ritalin’, *Science in Context* 15 (2002), 577-603, at p. 593. Presumably due to the influence of a psychoanalytically-oriented editorial board, and especially editor Eveleen Rexford, who ran the journal until 1976, *JAACP* refrained from running advertising until 1982. During that year the format of the journal changed considerably, incorporating not only advertising, but also new features such as a letters to the editor section. Perhaps delineating this change in philosophy, the revamped journal featured a special section on pharmacotherapy of children, focusing primarily on the use of drugs to treat hyperactivity. In contrast, the *International Journal of Social Psychiatry* had begun selling advertising space to drug companies as early as their third volume in 1957/1958.

⁵⁵⁹ Nancy Tomes, ‘The Great American Medicine Show Revisited’, *Bulletin of the History of Medicine* 79 (2005), 627-63, at p. 635.

of blaming parents, and especially mothers, for their children's mental health problems. As sociologist Ilina Singh has noted, 'weary of mother-blame ... for mothers with problem boys, the news about drug treatment and the emphasis on the organic nature of children's behavior problems appears to have been very welcome'.⁵⁶⁰ Shula Edelkind, for example, was continually blamed for her son's behaviour problems. Her interactions with both mental health workers and people in her neighbourhood made her believe that, 'whatever is wrong with the kid, obviously, it's the mother's fault. She feels like a failure, like a bad parent.' But when Edelkind was first told that her son's problem was genetic, neurological and had little to do with her parenting skills, she 'was happy; I was off the hook, it wasn't my fault'.⁵⁶¹

Edelkind felt she was 'off the hook' in a different way when she was told by her son's physician that all that was needed to treat him was a pill, specifically Ritalin. Her first impression of Ritalin, like that of many parents, was that it 'was amazing. It was a wonder drug'.⁵⁶² Indeed, biological psychiatrists had an enormous advantage over their disciplinary rivals in that their method for treating hyperactivity could evoke immediate, and often dramatic, improvements in behaviour. As pioneering hyperactivity researcher Maurice Laufer stated in an interview for the *New York Times*, stimulant drugs provided psychiatrists with 'one of the few situations in which you can do something quickly for people'.⁵⁶³ Laufer's colleague, Eric Denhoff, was so impressed by the efficacy of stimulant drugs that he considered 'it as "sort of criminal"' to withhold treatment from those who can use it'.⁵⁶⁴

Although some biological psychiatrists were puzzled by the fact that stimulants seemed paradoxically to calm hyperactive children, the belief in their effectiveness was such that, in

⁵⁶⁰ Singh, 'Bad Boys, Good Mothers', 593.

⁵⁶¹ Shula Edelkind, Interview.

⁵⁶² Shula Edelkind, Interview.

⁵⁶³ Maurice Laufer quoted in Robert Reinhold, 'Drugs that Help Control the Unruly Child', *New York Times*, 5 July 1970, 96.

⁵⁶⁴ Eric Denhoff quoted in Reinhold, 'Drugs that Help', 96.

some cases, stimulants were used as a diagnostic tool: if they calmed down an overactive, impulsive child, then the child likely had hyperactivity.⁵⁶⁵ More importantly for parents, however, was the fact that ensuring that their hyperactive children had their medicine was a quicker, easier and less expensive treatment modality than arranging for psychotherapy or analysing and attempting to change the social factors that might be contributing to such behaviour. Unlike other psychiatric approaches to hyperactivity, biological psychiatrists appeared to be able to establish the efficacy of Ritalin, and for many parents of hyperactive children, such drugs seemed to be veritable magic bullets.

Despite the success of biological psychiatrists in promoting a genetic, neurological understanding of hyperactivity, as well as pharmaceutical treatment of the disorder, there were enough gaps in their explanation of hyperactivity and concerns about their treatment practices for Feingold's idea to gain considerable attention. Ironically, the chief argument against the biological method of treating hyperactivity involved precisely what had made their approach so popular, namely, the use of stimulants to treat hyperactive children.

According to *New York Times* columnist Jane E. Brody, 'many parents dislike the idea of giving their children a potent drug day after day and are readily attracted to seemingly safer therapies, such as the diet Dr. Feingold has devised'.⁵⁶⁶ Indeed, interviews with parents who used the Feingold diet to treat their hyperactive children have demonstrated that fears about the side-effects of Ritalin, as well as overall uneasiness about using drugs to treat

⁵⁶⁵ Anonymous, 'Drugs Seem to Help Hyperactive Children', *JAMA* 214 (1970), 2260-2, at p. 2262; Larry B. Silver, 'The Playroom Diagnostic Evaluation of Children with Neurologically Based Learning Disabilities', *Journal of the American Academy of Child Psychiatry* 15 (1976), 240-56, at p. 253; Leighton Y. Huey Mark Zetin, David S. Janowsky and Lewis L. Judd, 'Adult Minimal Brain Dysfunction and Schizophrenia', *American Journal of Psychiatry* 134 (1977), 1563-5; Werry, 'The Use of Psychotropic Drugs', 453

⁵⁶⁶ Jane E. Brody, 'If the Child Seems to be "Bad," He Could Have Hyperkinesis', *New York Times*, 1 December 1976, 63.

misbehaviour, were among the most important factors leading to their employment of the Feingold diet.⁵⁶⁷

As Sean Corr, a father from Rhode Island explained: ‘I know medication works for a lot of people, but it didn’t seem like the thing to do to take a 6-year-old kid and give him Ritalin or something of that nature, you know, and drug him up to behave the way you want him to behave.’⁵⁶⁸ Sean’s wife, Carrie Corr, added that she was also concerned that Ritalin might affect her son’s growth, and that he would use it as ‘a crutch’, avoiding responsibility for his behaviour.⁵⁶⁹ Susan Leitner was also worried that Ritalin could suppress her son’s appetite, but also recalled news stories about amphetamine abuse that described ‘Ritalin being like an upper for regular folk’. The combination of these factors made her ‘dead set against Ritalin’.⁵⁷⁰ Other parents, such as Marta Phillips of Portland, Oregon, concurred, stating that hyperactivity ‘is probably what he’ll always have and he needs to learn to deal with it. He needs to learn to recognise it. He needs to learn to control himself. He needs to learn. . . . A pill doesn’t teach you that.’ Phillips added, however, that medication had value as a last resort.⁵⁷¹ Finally, Lora Hollins, a Michigan mother, emphasised that her experience working with psychiatric patients and participating in clinical trials as a mental health worker meant that she ‘certainly wasn’t going to put my child on Ritalin’.⁵⁷²

Some parents were inclined to try drugs, but abandoned them due to side effects or ineffectiveness.⁵⁷³ Texan Taunya Stevenson found that Ritalin made her five-year-old son Joshua ‘even more aggressive and . . . exasperated . . . He started cursing quite a lot, . . . and I

⁵⁶⁷ Bonnie Kowaliuk; Sean Corr, In-Person Interview, 6 December 2007; Lora Hollins, Telephone Interview, 17 February, 2008.; Anonymous, Telephone Interview, 30 January 2008.

⁵⁶⁸ Sean Corr, Interview.

⁵⁶⁹ Carrie Corr, Email Interview, 20 January 2008.

⁵⁷⁰ Susan Leitner, Telephone Interview, 8 April 2008.

⁵⁷¹ Marta Phillips, Telephone Interview, 13 February 2008.

⁵⁷² Lora Hollins, Interview.

⁵⁷³ Shula Edelkind; Anonymous, Email Interview, 29 January 2008; Colleen Davis.

wasn't like that. I don't know where all that came from.⁵⁷⁴ In order to deal with Joshua's hyperactivity, but also his depression and other psychological problems, his physician had him taking up to three different medications, totalling nine pills per day. Not wanting her son on so many medications, Taunya sought the opinions of other physicians, but they all tried to convince her that finding that right combination of drugs or getting the dosage adjusted would be the key.⁵⁷⁵

Although Shula Edelkind was impressed with some of the drugs prescribed to her son, she soon found that:

after a little while they couldn't get it right. He was either a zombie or as it wore off he was crazy and throwing furniture. And I remember thinking, I'm so glad he's small for his age. And he was small, he wasn't growing on Ritalin, either. ... And the neurologist said - we went to the neurologists every month, very faithfully - and he said, 'Oh it's okay that he's not growing, I'll give him growth hormones.' ... They changed his medication to Cylert and that was another wonder drug. It really kept the lid on. He was not the same child anymore. He was quiet ... [but] he was hallucinating. It's hard to know if you're child's hallucinating when they're very young and they're not communicating very well. ... Well, he was hallucinating and eventually I understood it because he told me, 'You know today my teacher's tongue was long and green and furry.'

Edelkind's son also became cognizant of the side effects, and by the time he was nine-years-old, expressed to his mother that he did not 'want anybody to mess with my brain anymore'. Shortly after she took him off all medication.⁵⁷⁶

Although one of the reasons Ritalin became preferred over stronger amphetamines and tranquilisers was that it was less dangerous, the drug nevertheless boasted its share of

⁵⁷⁴ Taunya Stevenson, Telephone Interview, 29 March 2008.

⁵⁷⁵ Tammy Frankenberger, 'Correspondence to Oprah Winfrey', July 2005. This correspondence was a letter written by Taunya Stevenson's sister to the talk show host Oprah Winfrey in the hopes of getting Taunya and her son on the Oprah Winfrey Show. They have not been on the show as of yet.

⁵⁷⁶ Shula Edelkind, Interview.

worrying side effects. As Edelkind described, one of these side effects was inhibited growth, but parents were also warned about insomnia, anorexia, irritability, heart rate changes, hallucinations and unknown long-term effects.⁵⁷⁷ Concern about side effects was so acute that a number of articles were published during the mid-1970s which advocated prescribing hyperactive children caffeine, instead of Ritalin. The chief proponent of this alternative, psychiatrist Robert Schnackenberg, observed that many of his hyperactive patients self-medicated with coffee which, he noted, was cheaper and less controversial than Ritalin.⁵⁷⁸ Although some researchers replicated Schnackenberg's findings, most found that caffeine was not as effective as Ritalin.⁵⁷⁹

While physicians could read about the side effects of Ritalin in medical journals, parents learned about such drugs from the media. American newspapers began reporting on the increasing numbers of prescriptions for Ritalin and other hyperactivity drugs as early as the mid-1960s. Although newspaper stories differed with regards to whether stimulant drugs

⁵⁷⁷ Although problems with growth, appetite and sleeping patterns were the most commonly cited side effects, in one case study hallucinations associated with Ritalin use were so 'severe, dramatic, and very frightening to the families it was not considered ethical to attempt replication'. Alexander R. Lucas and Morris Weiss, 'Methylphenidate Hallucinosis', *JAMA* 217 (1971), 1081-91, at p. 1081.

⁵⁷⁸ Schnackenberg also observed that countries where caffeinated beverages are not usually consumed by children, for example, the United States and Canada, had higher rates of hyperactivity than countries where children regularly consumed such beverages, such as in South America. Although social factors have likely had much more to do with this phenomenon - and South America has since 'caught up' with North America with regards to hyperactivity diagnoses - the author witnessed cases in which adolescents diagnosed with hyperactivity have 'self-medicated'. In one instance, a young man took a two litre bottle of Pepsi Cola with him everywhere in case he needed a 'fix'. Although he did not want to go onto Ritalin, one of the health professionals helping him urged him to do so because of concerns about the amount of sugar and chemicals he was consuming. Another young man would consume a one litre pot of coffee every morning when he awoke. Impatient for this to brew, he would drink a cup of instant coffee while he waited. Robert Schnackenberg, 'Caffeine as a Substitute for Schedule II Stimulants in Hyperkinetic Children', *American Journal of Psychiatry* 130 (1973), 796-8; David Pineda, Alfredo Ardila, Monica Rosselli, Beatriz E. Arias, Gloria C. Henao, Luisa F. Gomez, Sylvia E. Mejia, Martha L. Miranda, 'Prevalence of Attention-Deficit/Hyperactivity Disorder Symptoms in 4- to 17-Year-Old Children in the General Population', *Journal of Abnormal Child Psychology* 27 (1999), 455-62.

⁵⁷⁹ Frederic T. Zimmerman and Bessie B. Buregemeister, 'Action of Methyl-Phenidylacetate (Ritalin) and Reserpine in Behavior Disorders of Children and Adults', *American Journal of Psychiatry* 115 (1959), 323-8; Barry D. Garfinkel, Christopher D. Webster and Leon Sloman, 'Methylphenidate and Caffeine in the Treatment of Children with Minimal Brain Dysfunction', *American Journal of Psychiatry* 131 (1974), 723-8; Robert D. Huestis, L. Eugene Arnold and Donald J. Smeltzer, 'Caffeine Versus Methylphenidate and d-Amphetamine in Minimal Brain Dysfunction: A Double-Blind Comparison', *American Journal of Psychiatry* 132 (1975), 868-70; Philip Firestone, Jean Davey, John T. Goodman and Susan Peters, 'The Effects of Caffeine and Methylphenidate on Hyperactive Children', *Journal of the American Academy of Child Psychiatry* 17 (1978), 445-56.

were positive or negative, nearly all of them stressed that such drugs were controversial.⁵⁸⁰

Parents who read such stories, therefore, were aware of the debate surrounding the efficacy, safety and ethics of prescribing drugs to hyperactive children, and, ultimately, their decision to fear or embrace Ritalin rested on how they weighed the arguments made by both the drug's supporters and critics.

As *New York Times* science columnist Robert Reinhold (1941-1996) described, 'the increasing use of drugs to help children with learning disabilities is generating a sharp controversy among medical men'.⁵⁸¹ Reinhold proceeded to quote physicians who debated, for example, over whether the long-term effects of hyperactivity drugs should be considered or not. While paediatrician Sidney J. Adler from Orange County, California, admitted, 'I don't know what the drug will do in twenty years ... but I have to try to do what we can do now to keep the kid from winding up in juvenile hall', Richard D. Young, a psychology professor at Indiana University, stated that 'I shudder when I hear my colleagues suggest you can go ahead and give drugs to children ... We really don't know what are the effects of a lot of these drugs on a lot of processes over the long run'.⁵⁸² The headline of the story Reinhold wrote two years later, 'Drugs Seem to Help Hyperactive Children', suggested that the reporter had come to a conclusion regarding the debate, but he nonetheless continued to emphasise that the issue was controversial. He warned, for example, that 'the treatment often involves keeping the children on amphetamines, which are widely abused, for many years.

⁵⁸⁰ In contrast, Singh notes that none of the parenting magazines she analysed during the period 1945-1965 (*Woman's Day* and *Parents*) 'contained an overtly negative article on the issues surrounding children and psychostimulant medication'. She suspects that one reason for this was that such 'magazines did not give voice to dissent during this period' and that hyperactivity was perceived as a biological problem requiring a biological solution. Singh's observations, however, are not representative of the controversy expressed in both medical and popular literature during the early 1960s, a period she does cover, or the late 1960s and early 1970s, which she does not address. Singh, 'Bad Boys, Good Mothers', 594-5.

⁵⁸¹ Robert Reinhold, 'Learning Parley Divided on Drugs', *New York Times*, 6 February 1968, 40.

⁵⁸² *Ibid.*, 40.

This has led to fears of long-term damage and charges that youngsters were being drugged into submission.⁵⁸³ In another article which also superficially supported the prescribing of Ritalin, Reinhold again mentioned the controversy surrounding the practice and reported on ‘children swapping their pills in the school yard with unfortunate effects’.⁵⁸⁴

Reinhold’s comment that amphetamines had been ‘widely abused’ highlighted one of the other primary reasons why many parents were wary about hyperactivity drugs. Ritalin, which had largely replaced the use of more powerful amphetamines such as Dexadrine by the late 1960s, was nevertheless linked in a number of ways to a wide range of illegal amphetamines known generally as ‘speed’, but also by the street names “‘splash”, “‘crank”, “‘rhythm”, “‘meth” or “‘crystal””.⁵⁸⁵ According to alarming reports, such as a *New York Times* story entitled ‘The Speed that Kills’, the ‘cannibalism of speed’ had transformed ‘quiet flower children [into] ravaged scarecrows’ or ‘speed freaks’, whose high risk behaviour could lead to violence, suicide and health problems such as ‘colds, infections, muscle tremors, cardiac problems, nausea, cramps, respiratory problems and hepatitis’.⁵⁸⁶ Such concerns in Sweden led to a complete ban on amphetamines, including Ritalin, during the late 1960s.⁵⁸⁷

The gateway to abusing such drugs was often connected with attempts to improve academic performance. The opening paragraphs of ‘The Speed that Kills’, for example, focussed on a college student who, facing a deadline, accepted his girlfriend’s offer of some little yellow diet pills which were mild amphetamines:

⁵⁸³ Reinhold, ‘Drugs that Help’, 96.

⁵⁸⁴ Robert Reinhold, ‘Rx for Children’s Learning Malady’, *New York Times*, 3 July 1970, 27.

⁵⁸⁵ Jonathan Black, ‘The Speed that Kills’, *New York Times*, 21 June 1970, 15, 18, 22-5, at p. 15. For more on the history of amphetamines, see: Leslie Iverson, *Speed, Ecstasy, Ritalin: The Science of Amphetamines* (Oxford: Oxford University Press, [2006] 2008); Nicolas Rasmussen, *On Speed: The Many Lives of Amphetamine* (New York: New York University Press, 2008).

⁵⁸⁶ Black, ‘The Speed that Kills’, 15, 22.

⁵⁸⁷ Constance Holden, ‘Amphetamines: Tighter Controls on the Horizon’, *Science* 194 (1976), 1027-8.

With the first pill, Norman's mind clicked into gear and his fingers pattered over the keyboard as intricate insights streamed out of his head. After 10 hours he took a break and cleaned out all the drawers of his desk, arranged the pens and pencils in precise parallels, and stacked all his books so that the bottom corners were exactly even. Then he slid the pile so it coincided perfectly with the right angle at the corner of his desk. He stared at the pile for 20 minutes. Then he popped another pill, whistled through 10 more hours of typing and polished up the conclusion of his thesis with some more rather arcane insights. Norman drank half a quart of orange juice, emptied the icebox, and cleaned out all the shelves. Then he retyped some earlier pages which a dirty eraser had smudged, called up his girl and chattered gaily for 40 minutes. Then Norman passed out for 10 hours with dreams of an A in his head. He got a B-plus. Norman was speeding, but well under the limit.⁵⁸⁸

Although Norman's flirtation with speed might have seemed innocent enough, the euphoria associated with such drugs, reportedly to be 'potentially as addictive and debilitating as heroin', meant that such experiments could lead to abuse.⁵⁸⁹ In a subtler way, however, the scenario of a college student dabbling in amphetamines in order to finish an assignment was not so different than parents taking up a physician or teacher's advice to consider a prescription of Ritalin for their child. In both cases, academic difficulties could be allayed with the help of stimulant drugs. While some parents and students found the potential benefits of such assistance tempting, others were alarmed that it could lead to overdependence on medication and experimentation with other drugs. As child psychiatrist Mark Stewart described, 'by the time a child on drugs reaches puberty, he does not know what his undrugged personality is and, even worse, his family does not know how to accept it'.⁵⁹⁰

Not only did Ritalin share a chemical composition similar to more powerful amphetamines, it was also sold itself as a drug of abuse on the street. A Seattle health worker went as far as to say that Ritalin was 'the No. 1 drug-abuse problem in that city', and was responsible for

⁵⁸⁸ Black, 'The Speed that Kills', 15.

⁵⁸⁹ Boyce Rensberger, 'Doctors Consider Pledge to Curb Amphetamine Prescriptions', *New York Times*, 18 June 1971, 40.

⁵⁹⁰ Anonymous, 'Classroom Pushers', *Time*, 26 February 1973, www.time.com/time/magazine/article/0,9171,910580,00.html?promoid=googlep, accessed 23 March 2009.

‘severe medical problems including multiple abscesses, and damage to heart valves’ when it was dissolved and injected by addicts.⁵⁹¹ Although observers worried about Ritalin’s abuse as a street drug, its legitimate use also courted controversy during the early 1970s.⁵⁹² Of particular concern was the increase in stimulant prescriptions and the notion that teachers and other school officials were too eager to convince parents that medical intervention was required to improve their child’s school performance or behaviour. One story, for example, described a Rhode Island mother who felt she was ‘forced by school officials into drugging her child ... She said she had been constantly harassed by the school about her child’s behavior and got a note from the school nurse which stated simply: “Your child is hyperactive. He doesn’t sit still in school. Please see a physician.”’⁵⁹³ Another article reported a six-year-old girl in Michigan whose teacher convinced her father to consider Ritalin. The father reported that ‘the drug made her so withdrawn that sometimes she would sit for hours doing nothing’. After getting ‘panicky’ about such behaviour, he took her to a psychologist who determined that she ‘was perfectly healthy ... [and needed] drill in basic reading, not drugs’.⁵⁹⁴ These stories echo the experience of Taunya Stevenson who was told by her son’s private Christian school that unless her son was put on medicine, he could not come to school.⁵⁹⁵

Even more alarming than such individual anecdotes, however, were reports in 1970 that somewhere between five and ten per cent of the school-age population in Omaha, Nebraska,

⁵⁹¹ Harold M. Schmeck, Jr., ‘Tighter Controls Asked on 2 Drugs’, *New York Times*, 17 July 1971, 8.

⁵⁹² Ritalin is still sold as a street drug today. S. E. McCabe, John R. Knight, Christian J. Teter and Henry Wechsler, ‘Non-Medical Use of Prescription Stimulants Among US College Students: Prevalence and Correlates from a National Survey’, *Addiction* 100 (2005), 96-106.

⁵⁹³ Randall Richard, ‘Drugs for Children – Miracle or Nightmare?’, *The Providence Journal*, 8 February 1972, 1.

⁵⁹⁴ Anonymous, ‘Classroom Pushers’.

⁵⁹⁵ Ironically, Stevenson had taken her son out of a previous private Christian school because they insisted that she employ corporal punishment. As she described, ‘they would not let [us] stay [at the school] unless I paddled him ... so he was being spanked a lot and I really think that when you add the low self esteem to whatever other problems he had that does more damage than anything’. Taunya Stevenson.

or 62 000 children, was on drugs for behaviour and learning problems.⁵⁹⁶ The story, reported on the front page of the *Washington Post*, prompted not only a national outcry, but Congressional hearings in 1970, led by New Jersey Congressman Cornelius E. Gallagher, and a 1971 conference on the subject organised by the Office of Child Development.⁵⁹⁷ Although the 1971 conference would tacitly approve the prescription of stimulants for hyperactivity, provided parents were not coerced into agreeing to such measures and long-term follow-up was conducted, the mixed messages about Ritalin in the media did much to counter the glowing reports of its efficacy found in medical journals.⁵⁹⁸ Although some Ritalin advocates, such as pioneering hyperactivity researcher Eric Denhoff, were describing the drug in 1970 as ‘the penicillin of children with learning disabilities’, to parents it might have seemed less like a magic bullet and more like a form of ‘black magic’, a drug whose benefits might not outweigh its costs.⁵⁹⁹

Conclusion

The history of American psychiatry and hyperactivity demonstrates that Feingold’s hypothesis regarding hyperactivity was one of many which contended for legitimacy during the 1960s and 1970s. Although some psychiatrists, such as Leon Eisenberg, favoured a more pluralistic approach, by the publication of *Why Your Child is Hyperactive*, biological interpretations and pharmaceutical treatments of hyperactivity prevailed, and most of what social psychiatrists and psychoanalysts had to say about the disorder had been either rejected or disregarded. The resolution of such debates occurred not because the biological approach

⁵⁹⁶ Robert Maynard, ‘Omaha Pupils Given “Behavior” Drugs’, *The Washington Post*, 29 June 1970, A1; Lawrence Diller, ‘The Run on Ritalin: Attention Deficit Disorder and Stimulant Treatment in the 1990s’, *Hastings Center Report* 26 (1996), 12-18, at p. 12.

⁵⁹⁷ Peter Conrad, ‘The Discovery of Hyperkinesia: Notes on the Medicalization of Deviant Behavior’, *Social Problems* 23 (1975), 12-21, at p. 15.

⁵⁹⁸ *Ibid.*

⁵⁹⁹ Also like penicillin, according to Denhoff, was the over-prescription of Ritalin. In an interview with *Time* magazine in 1973 Denhoff warned that half of the children on Ritalin in Rhode Island should not have been on the drug. Reinhold, ‘Rx for Children’s Learning Malady’, 27; Denhoff quoted in Anonymous, ‘Classroom Pushers’.

was necessarily more valid, but rather because it was more direct, facile and inexpensive, and because it capitalised on contemporary developments in psychopharmaceutical research. Nevertheless, none of the psychiatric approaches to hyperactivity, especially when viewed singularly, escaped criticism.

The history of hyperactivity and American psychiatry during the 1960s and early 1970s also suggests that understanding the history of psychiatry during this period was more complicated than some historians have assumed. In general, historians have overlooked the ideological divisions that separated psychiatry and have failed to recognise the belief of many contemporary psychiatrists, physicians and academics that mental illness was one of the greatest threats faced by the United States. While David Healy has outlined the ideological differences between psychoanalysts and biological psychiatrists during the 1960s, for example, he has under-estimated the popularity of social psychiatry and the demand for preventative psychiatry.⁶⁰⁰

Other historians have over-simplified the relationship between broad social trends during the post-war period and the provision and acceptance of psychiatric treatment. David Herzberg and Jonathan Metzl have stressed how tranquilisers were used to reinforce gender stereotypes, but others, including Ali Haggett, have argued that such treatment could also give women 'clarity of thought and an opportunity to assess their life circumstances with a view to change'.⁶⁰¹ Similarly, many sociological explanations for why hyperactivity became so prominent during the 1960s and 1970s have failed to address why parents became

⁶⁰⁰ Healy, *The Antidepressant Era*, 219-20.

⁶⁰¹ Ali Haggett, 'Housewives, Neuroses, and the Domestic Environment in Britain, 1945-70' in Mark Jackson (ed.), *Health and the Modern Home* (New York: Routledge, 2007), 84-110, at p. 98; Jonathan Michel Metzl, *Prozac on the Couch: Prescribing Gender in the Era of Wonder Drugs* (Durham, NC: Duke University Press, 2003), 5-10; David Herzberg, *Happy Pills in America: From Miltown to Prozac* (Baltimore: Johns Hopkins University Press, 2009), 47-82.

convinced that their children were hyperactive and required medical treatment.⁶⁰² Metzl's broader argument that Freudian thought remained entrenched in American psychiatry is also unsupported by the history of hyperactivity. In contrast, one of the reasons neurological explanations for hyperactivity became so popular was that they did not blame parenting skills or family dynamics for the disorder.

The complex manner in which conceptualisations of hyperactivity were developed, debated and either accepted or rejected highlights not only the contentious nature of psychiatric knowledge, but also how explanations of mental illness could be open to interpretation.

When it came to the biological approach to hyperactivity, for example, parents who favoured the Feingold diet found themselves in a difficult position. While they accepted the premise that hyperactivity had something to do with the nervous system, they rejected the pharmacological treatment of the disorder. Although most parents with hyperactive children during the 1970s ignored the warnings about Ritalin and, instead, concentrated on the positive effect it seemed to have, others were either not willing to do so, or found that drugs were not a satisfactory intervention. Often desperate for any form of succour, these families turned to the Feingold diet.

Feingold's specific explanation for hyperactivity also capitalised on the aetiological shortcomings of biological psychiatry. Unlike biological psychiatrists, whose vague explanations implied that some kind of imprecise genetic defect was at fault, Feingold delineated a clear causation for hyperactivity, one which he claimed to be able to prove by 'turning the disorder on and off' with challenges of food additives.⁶⁰³ Feingold's explanation

⁶⁰² Peter Schrag and Diane Divoky, *The Myth of the Hyperactive Child: And Other Means of Child Control* (New York: Penguin Books, [1975] 1982); Peter Conrad, *Identifying Hyperactive Children: The Medicalization of Deviant Behavior* (Toronto: Lexington Books, 1976); Rick Mayes and Adam Rafalovich, 'Suffer the Restless Children: The Evolution of ADHD and Paediatric Stimulant Use, 1900-1980', *History of Psychiatry* 18 (2007), 435-57.

⁶⁰³ Feingold, *Why Your Child is Hyperactive*, 34.

attracted parents to his diet not only because it gave them a tangible cause against which they could take action, but also because, as the next chapter argues, it tapped into contemporary fears about the chemicalisation of the food supply and broader concerns about the effects of a wide array of pollutants on human health.

Chapter 7

Concerns about Chemicals: 'Food just isn't what it used to be'

The role of chemicals in the food supply was an issue that caused enormous debate during the post-war period and divided opinion not only about food, but also about technology, modern lifestyles and the aetiology of disease. While some, such as English psychiatrist Richard Mackarness, advocated a return not only to a chemical-free diet, but to a ‘stone-age diet’ based on protein rather than carbohydrates, others, such as nutritionist Frederick Stare and epidemiologist Elizabeth Whalen, believed that food additives were of enormous benefit and a sign of progress.⁶⁰⁴ By the emergence of the Feingold diet, most Americans would have agreed with journalist Jacquin Sanders that ‘food just isn’t what it used to be’, but not all would have agreed that the changes had been detrimental to the American diet.⁶⁰⁵ As this chapter suggests, such divisions accentuated the controversy surrounding the Feingold diet in both the public press and medical literature.

For parents who already believed that food additives were unhealthy and had either made or considered making their diet more organic, success with the Feingold diet crystallised many of their ideas about food which had been forming for a number of years. In contrast, other parents came to the Feingold diet from either the other side of the debate over food additives, believing that there was nothing wrong with such chemicals, or without any clear sense that such debates were occurring. Once these parents began investigating Feingold’s theory, however, they tapped into a vast array of literature, dating primarily to the late 1960s and early 1970s, but also prior to that, which claimed that the modern American diet was unhealthy. On the one hand, this legacy of suspicion helped to legitimise the Feingold diet for parents who might otherwise have questioned how food additives could cause behavioural disorders. On the other hand, the divisive nature of debates surrounding food additives meant that, no matter how Feingold distanced himself from such critics and attempted to make his

⁶⁰⁴ Elizabeth M. Whalen and Frederick J. Stare, *Panic in the Pantry: Food Facts, Fads and Fallacies* (New York: Atheneum, 1975); Richard Mackarness, *Not All in the Mind: How Unsuspected Food Allergy Can Affect Your Body and Your Mind* (London: Pan Books, 1976).

⁶⁰⁵ Jacquin Sanders, ‘Organic Food: A Growing Market’, *Washington Post*, 28 June 1970, 103.

diet appear scientifically valid, it was perhaps inevitable that it would be perceived as being on the radical side of the debate.

Although Feingold linked the rise in hyperactivity to post-war changes in how food was produced, packaged and preserved, there had been concerns about food adulteration and food safety well before the emergence of TV-dinners and microwavable meals. As historian Harvey Levenstein indicates, processed food products began arriving in American stores during the 1880s and, within two decades, spurred on by the publication of Upton Sinclair's *The Jungle* (1906), American journalists were warning their readers about food additives.⁶⁰⁶ Sinclair's novel, which described the deplorable working and sanitary conditions in Chicago meat-packing plants, contributed to the passage of the Pure Food and Drug Act of 1906, which was intended to prevent 'the manufacture, sale, or transportation of adulterated or misbranded or poisonous or deleterious foods, drugs, medicines, and liquors'.⁶⁰⁷

There was a key difference, however, in the adulteration targeted by legislation such as the 1906 Act, and that which attracted attention during the 1960s. The adulteration described in *The Jungle* and other publications, which criticised the increasingly industrialised food industry, tended to focus on adding inedible or unsanitary substances to food in order to improve profit margin. For example, an anonymous physician writing in 1885 warned of the 'man who willfully adds a non poisonous substance to an article which he sells, for the sake

⁶⁰⁶ Harvey Levenstein, *Revolution at the Table: The Transformation of the American Diet* (Oxford: Oxford University Press, 1988), 39. Historian Derek Oddy places similar developments in Britain at roughly the same time, in the 1890s. In Britain, too, 'chemical preservatives, such as borax or formalin, were used extensively in foodstuffs to extend shelf life. The opportunities for adulteration and the use of additives and improver was irresistible, and there were some notable instances when consumers' health was seriously affected as producers cut corners.' Derek J. Oddy, *From Plain Fare to Fusion Food: The British Diet from the 1890s to the 1990s* (Woodbridge: The Boydell Press, 2003), ix, 31.

⁶⁰⁷ Federal Food and Drugs Act of 1906 (The Wiley Act), Public Law Number 59-384, 30 June 1906. Sinclair sent a copy of *The Jungle* to President Theodore Roosevelt who sent two commissioners to investigate the author's claims. Despite the fact that the packers were given two weeks notice of the commissioners' visit, nearly all of Sinclair's allegations were verified. Upton Sinclair, *The Jungle* (Harmondsworth: Penguin, [1906] 1974). James Harvey Young, 'The Pig that Fell into the Privy: Upton Sinclair's *The Jungle* and the Meat Inspection Amendments of 1906', *Bulletin of the History of Medicine* 59 (1985), 467-80, at pp. 468-70.

of increasing its bulk or weight, and afterward retails that to his customers as pure', as well as the 'man who adds that to his goods which shall injure the health of the partaker'. Such practices, according to the writer, 'greatly effects the public health, and ... thousands annually owe their deaths to the tricks of the trade'.⁶⁰⁸

Although the introduction of chemicals into the food supply following the Second World War also had much to do with profit margin, the difference was that post-war food additives were there purposefully to enhance flavour, retain colour and preserve, as well as to facilitate the invention of a whole new range of food products and fast food restaurants in which such products were sold.⁶⁰⁹ Moreover, as Levenstein discusses:

most American consumers were impressed by these achievements, and until well into the 1960s they showed little concern for the methods and ingredients which food processors employed to turn out a host of new products ... There was little inclination to question the products of the food business, which seemed to make life easier for the housewife with each new chemical breakthrough.⁶¹⁰

Such sentiments were echoed in FAUS director Jane Hersey's original impressions of processed food:

I was a modern little homemaker, I mean I did all ... I mean, people who made things from scratch to me were unbelievably out of touch with the times. Why should I do something when Betty Crocker had already done it for me. I thought ... I was living the American dream. ... There was a woman who used to write about how you could make your favourite desserts, but cut back on calories, and, you know, with fake this and fake that, I thought that was wonderful.⁶¹¹

The sales of processed foods such as Betty Crocker instant cake mixes and Swanson's TV Dinners were profitable not only because of their novelty, but also because they appeared to

⁶⁰⁸ Bread, for example, could be adulterated with 'chalk, pipe clay, plaster of paris, alum, carbonate of ammonia, sulphate of zinc'. Anonymous, 'Deleterious Food', *Brooklyn Daily Eagle*, 20 September 1885, 12.

⁶⁰⁹ John A. Jakle, 'Roadside Restaurants and Place-Product Packaging' in George O. Carney (ed.), *Fast Food, Stock Cars, and Rock 'n' Roll* (London: Rowman and Littlefield, 1995), 97-117, at p. 97.

⁶¹⁰ Levenstein, *Revolution at the Table*, 202.

⁶¹¹ Jane Hersey, Telephone Interview.

liberate housewives and working mothers from the drudgery of the kitchen. During the post-war period, most academic nutritionists and government agencies supported the proliferation of food additives.⁶¹² One of the manifestations of the tight relationship between academic nutritionists and the food industry was the creation of the Nutrition Foundation in 1941. Envisioned and funded by food producers, the Nutrition Foundation published an academic journal entitled *Nutrition Reviews*, which included articles by academic nutritionists and was edited by Harvard nutrition scientist Frederick Stare. According to Levenstein, the Nutrition Foundation was often ‘used to marshal scientific opinions to correct “superficial and faddish ideas” and to combat those questioning any of the 704 chemicals that by 1958 were commonly used in foods’.⁶¹³ Although such chemicals were seen by the Nutrition Foundation as improving the food supply, the potential danger posed by ingesting synthetic colours, flavours, preservatives and pesticides also prompted both considered and visceral reactions from many ecologists, politicians, journalists and physicians.

The beginning of this chapter traces two of the major developments that precipitated fears of food additives during the late 1960s, namely the passing of the Delaney Clause in 1958 and the publication of *Silent Spring* in 1962. It then explores the rise of the natural food movement in the late 1960s and examines the controversy that emerged about the threat to health posed by chemicals used in the food supply. The chapter concludes by contending that debates about food additives reflected a broader climate of suspicion in American society which, in turn, contributed to interest in the Feingold diet. Although most Americans trusted the food industry and the governmental organisations that regulated it, many others had lost faith in the food supply and were prepared to consider theories that blamed it for ill health.

⁶¹² Harvey Levenstein, *Paradox of Plenty: A Social History of Eating in Modern America* (Oxford: Oxford University Press, 1993), 113-4; 134-5.

⁶¹³ *Ibid.*, 112.

The Delaney Clause, *Silent Spring* and the Fear of Food Additives

Despite the influence of the Nutrition Foundation, and the proliferation of processed food, by the late 1950s and early 1960s faith in the American food supply was beginning to be shaken.⁶¹⁴ Ironically, this concern was partially due to a shift in U.S. government policy regarding nutrition and health. As academic nutritionist Marion Nestle describes, until the 1960s government policy regarding food consumption was concerned with preventing nutritional deficiencies which could lead to diseases such as rickets, pellagra and scurvy.⁶¹⁵ As such, government advice was for Americans to eat more calories, rather than fewer, and not to worry about restricting their intake of any particular foods. By the 1960s, however, amidst concerns about certain chronic diseases, including heart disease, cancer and diabetes, government opinion had shifted and now encouraged Americans to eat fewer calories. Americans were instructed to avoid certain foods, particularly those containing high levels of fat, cholesterol, sugar and salt, as well as alcohol.⁶¹⁶ In this way, food shifted from being a protection against disease to being a cause of disease. Moreover, if traditional foodstuffs, such as beef, butter and eggs, could be vilified by the new approach to nutrition, synthetically-produced food additives could also be questioned for their impact upon health.

If rising rates of chronic disease encouraged a shift in how nutritionists perceived food generally, two developments during the post-war period targeted food additives as being specifically harmful to health. These were the passing of the Delaney Clause in 1958, which affected the process by which food additives were approved by the FDA, and the publication of Rachel Carson's *Silent Spring* in 1962. The reception of both the Delaney Clause and *Silent Spring* also highlighted that the chemicals utilised in post-war food processing were a matter of intense debate. While consumer groups, environmental activists and concerned

⁶¹⁴ Ibid., 130.

⁶¹⁵ Marion Nestle, *Food Politics: How the Food Industry Influences Nutrition and Health* (Berkeley: University of California Press, 2002), 31-3.

⁶¹⁶ Ibid., 38-9.

physicians warned that such chemicals were a major threat to human health, food and chemical processing companies, the FDA and sceptical health professionals argued that such additives, in contrast, posed no harm, or in some cases, benefited health.

Although the Chemicals in Food Products hearings occurred during 1950 and 1951, the Food Additive Amendment, nicknamed the ‘Delaney Clause’ after chairman Congressman James Delaney, was not added to the Food, Drugs and Cosmetics Act of 1938 until 1958. The Delaney Clause specified ‘that no additive shall be deemed to be safe if it has been found to induce cancer when ingested by man or animal’.⁶¹⁷ Two years later such principles were re-applied in the Color Additive Act.⁶¹⁸ The fact that it took so long to pass the amendment suggests that there was little political will during the 1950s to examine, let alone curtail, chemicals entering the food supply.⁶¹⁹ Indeed, Delaney had difficulty recruiting scientists to testify when he first launched the hearings.⁶²⁰

Nevertheless, in 1958 the bill received support from two sources, one conventional and one unexpected. First, the National Cancer Institute, spearheaded by the work of controversial pathologist Wilhelm Hueper, reported that ‘a number of chemicals long used in food might cause cancer in humans’.⁶²¹ Unlike previous warnings from individual clinicians, this report from a national organisation received a great deal of press. According to Delaney who felt

⁶¹⁷ Quoted in Richard A. Merrill, ‘Food Safety Regulation: Reforming the Delaney Clause’, *Annual Review of Public Health* 18 (1997), 313-40, at p. 318.

⁶¹⁸ Merrill, ‘Food Safety Regulation’, 320.

⁶¹⁹ According to the Senate Labor and Public Welfare Committee, the members of which wrote the legislative history of the bill, the Delaney Clause was the result of ‘the most extensive and intensive hearings on legislative proposals in a particular field in which we have knowledge’. Quoted in Charles H. Blank, ‘The Delaney Clause: Technical Naïveté and Scientific Advocacy in the Formulation of Public Health Policies’, *California Law Review* 62 (1974), 1084-1120, at p. 1088.

⁶²⁰ Levenstein, *Paradox of Plenty*, 112.

⁶²¹ Hueper was controversial not least because of his criticism of government cancer policy which concentrated on reducing levels of tobacco use; Hueper believed that pollution reduction should be a larger part of the war on cancer. Hueper was also in close contact with Rachel Carson as she was writing *Silent Spring*. Levenstein, *Paradox of Plenty*, 133; Christopher Sellers, ‘Discovering Environmental Cancer: Wilhelm Hueper, Post-World War II Epidemiology, and the Vanishing Clinician’s Eye’, *American Journal of Public Health* 87 (1997), 1824-35, at p. 1832.

that his efforts to gain support for his bill were akin to ‘screaming at the wind’, more influential were the efforts of fading Hollywood actress and health food advocate Gloria Swanson (1899-1983), who urged the wives of numerous congressmen to convince their husbands to support the amendment.⁶²²

The Delaney Clause was divisive for a number of reasons. First, it posed a threat to the use of many food additives used in both agricultural production and food processing because the cost of proving that these additives were safe was believed to be onerous.⁶²³ Food industry representatives also disagreed that substances found to be carcinogenic in animals should be automatically thought to be carcinogenic in humans.⁶²⁴ Related to this was a debate regarding the point at which a substance should be classified as a carcinogen.⁶²⁵ Certain additives were found to be carcinogenic, but only when consumed by humans in extremely large amounts (see cranberry example below), or in combination with other substances.⁶²⁶ Finally, there was also the argument that certain substances, such as the artificial sweetener saccharin, which could be carcinogenic at high levels of consumption, could also be beneficial to health, since they could help individuals lose excess weight.⁶²⁷

Given these complaints, according to Thomas H. Jukes (1906-1999), a prominent Anglo-American biologist then working for the American Cyanamid Company, the Delaney Clause represented ‘a serious concern to all manufacturing groups concerned with chemicals which

⁶²² Delaney claimed that the ‘the chemical lobby spent \$90,000 to defeat’ him in the 1956 congressional election. He won by only 45 votes when he normally won by a margin of two or three to one. Richard D. Lyons, ‘Congressman Says Actress’s Speech Helped Bar Cyclamates’, *New York Times*, 22 October 1969, 26.

⁶²³ If a pesticide left a residue on produce which was then meant to be consumed, it was considered a food additive. Levenstein, *Paradox of Plenty*, 134.

⁶²⁴ *Ibid.*, 134.

⁶²⁵ Beatrice Trum Hunter, *The Mirage of Safety: Food Additives and Federal Policy* (New York: Scribner, [1975] 1982), 160.

⁶²⁶ Historical trends in determining carcinogenicity have been explored by Christopher Sellers. For instance, the establishment of tobacco as an carcinogen made it difficult to determine if certain workplace chemicals, such as asbestos, were also causing cancer because so many workers smoked. Sellers, ‘Discovering Environmental Cancer’, 1832-3.

⁶²⁷ Anonymous, ‘The Case of the Useful Carcinogen’, *New York Times*, 7 December 1978, A22.

come into contact with food'.⁶²⁸ Confirming such fears, the Delaney Clause was invoked soon after its passage in the case of the chemical aminotriazole, a herbicide used in cranberry bogs.⁶²⁹ Although the FDA had banned the use of the herbicide in May 1959 because it had been shown to be carcinogenic, it was discovered nevertheless to have been used in a proportion of that year's crop. The contaminated cranberries were taken off the market, with the assurance that even if some of the berries made it into Thanksgiving dinner, people would have to consume fifteen thousand pounds of them to suffer any harm.⁶³⁰ Nevertheless, Americans refused to buy cranberries that November and the government eventually reimbursed producers the 8.5 million dollars lost due to the scare.⁶³¹

The fear of carcinogens also contributed to the impact of Rachel Carson's *Silent Spring*. This was partly due to the book's content, particularly its chapter 'One in Every Four', which linked environmental pollutants to cancer, but also to the fact that Carson spent much of her later years battling the disease, succumbing to a cancer-related heart attack in 1964.⁶³² But despite the unquestionable influence of *Silent Spring* in sparking environmental awareness and activism in the United States, Carson's book, like Delaney's amendment, was also contentious. *Silent Spring* was, on the surface, about the broad environmental dangers and health problems caused by the overuse of pesticides. Underlying the sordid story of pesticide use, however, was a message about ecology, and how plants, animals and humans were

⁶²⁸ Thomas H. Jukes, 'Food Additives', *Science* 134 (1961), 798.

⁶²⁹ William H. Rodgers, Jr., 'The Persistent Problem of the Persistent Pesticides: A Lesson in Environmental Law', *Columbia Law Review* 70 (1970), 567-611, at p. 593.

⁶³⁰ Levenstein, *Paradox of Plenty*, 134

⁶³¹ Rodgers, Jr., 'A Lesson in Environmental Law', 594.

⁶³² Rachel Carson, *Silent Spring* (London: Folio Society, [1962] 2000), 209-29; Linda Lear, *Rachel Carson: Witness for Nature* (London: Allen Lane The Penguin Press, 1998), 480. Philosopher and physicist Sheldon Krimsky has suggested that although 'cancer is certainly not the only adverse health effect of industrial chemicals ... it has largely eclipsed other diseases and reproductive effects as an object of public concern and scientific research'. He proceeds to suggest, however, that 'in the last few years, a new theory of environmental disease has emerged that explores a variety of human and animal abnormalities that are not explained by or investigated within the dominant cancer paradigm'. The possibility that environmental chemicals may interfere with the body's hormones is central to this new area of research. Sheldon Krimsky, *Hormonal Chaos: The Scientific and Social Origins of the Environmental Endocrine Hypothesis* (Baltimore: The Johns Hopkins University Press, 2000), 2.

bound symbiotically to each other and the state of the environment. The songbirds rendered silent by pesticide use were a tragedy unto themselves, but also a warning about what was in store for humans if the wanton use of DDT and other chemicals went unchecked.

While Carson's ability to describe chemical and ecological phenomena in a lyrical, poignant, yet scientifically rigorous manner captivated countless readers, it also threatened the chemical industry and the scientists and politicians allied to it. The ensuing debate was played out not only in industry journals, but also in the media, as both scientists and members of the media argued about Carson's claims. At the heart of the dispute was where to strike the balance between agricultural development, with the resulting availability of inexpensive food, and environmental stewardship. For example, United States Department of Agriculture (USDA) spokesman Ernest G. Moore, interviewed in the *Washington Daily News*, suggested that Americans were unwilling to return to life without pesticides: 'the balance of nature is a wonderful thing for people who sit back and write books or want to go out to Walden Pond and live as Thoreau did. But I don't know of a housewife today who will buy the type of wormy apples we had before pesticides.'⁶³³

Although the issue of how to mediate between agricultural development and environmental sustainability was perceived to be a crucial debate that had global implications, much of the criticism centred not on Carson's scientific arguments, but upon her manner of presenting it and whether or not she had the expertise to discuss the topic at all. According to her biographer, Linda Lear, Carson 'deliberately employed the rhetoric of the Cold War and the tone of moral crisis to persuade her readers of the urgency of her message'.⁶³⁴ Such tactics, though helping to propel *Silent Spring* to the top of the *New York Times* bestseller list during

⁶³³ Ernest G. Moore quoted in Lear, *Witness for Nature*, 413.

⁶³⁴ Lear, *Witness for Nature*, 428.

late 1962, resulted in a virulent response mounted in ‘equally ideological terms’.⁶³⁵ As Lear suggests, these criticisms tended to focus on three factors, unrelated to her ecological argument. These included the fact that she had written for a public, rather than a scientific, audience, her relative lack of scientific training - although having a master’s degree in zoology, she had never published in peer-reviewed journals or held an academic post - and her status as an unmarried woman. Carson was branded as an overly emotional, manipulative spinster whose ‘reason had been sacrificed to sentiment’.⁶³⁶

In many ways, these criticisms foreshadowed those directed at Feingold a decade later. Feingold, too, was accused of writing for a popular audience. Although he was a qualified physician, he was perceived as a clinician, rather than a medical researcher, and was criticised for reporting his clinical observations, rather than submitting his hypothesis to large double-blind clinical trials. Although Feingold’s gender was not questioned, his age was raised as a complicating factor. Specifically, Feingold’s detractors claimed that his grandfatherly charm could unduly influence parents’ assessments of the Feingold diet.⁶³⁷

Perhaps the most striking synthesis of the criticisms levelled against Carson was agricultural company Monsanto’s sarcastic parody of *Silent Spring*’s opening lines which they sent to newspapers across the United States:

Quietly, then, the desolate year began. Not many people seemed aware of the danger. ... How could the good life depend on something so seemingly trivial as bug spray? Where were the bugs anyway? The bugs were everywhere. Unseen. Unheard. Unbelievably universal. Beneath the ground, beneath the waters, on and in limbs and twigs and stalks, under rocks, inside trees and animals and other insects – and, yes, inside man.⁶³⁸

⁶³⁵ Ibid., 426-8.

⁶³⁶ Ibid., 430.

⁶³⁷ Anonymous, ‘Diet and Hyperactivity: Any Connection?’ *Nutrition Reviews* 34 (1976), 151-8.

⁶³⁸ The pages to which the Monsanto parody refers are found in Carson’s first chapter, ‘A Fable for Tomorrow’. Carson, *Silent Spring*, 39-40; Monsanto quoted in Lear, *Witness to Nature*, 430.

Time's anonymous reviewer of *Silent Spring*, while less creative, also focussed on these themes, stating that 'Miss Carson's ... emotional and inaccurate outburst in *Silent Spring* may do harm by alarming the non-technical public, while doing no good for the things that she loves'.⁶³⁹ Emphasis on 'Miss' Carson's gender, emotionality, scientific qualifications and prose style was also a hallmark of attacks launched on her by the Nutrition Foundation, which would later target Feingold, and other industry groups such as the National Agricultural Chemicals Association, the Manufacturing Chemists Association and the National Pest Control Association.⁶⁴⁰

Despite such attacks, the American public was willing to consider Carson's claims, and millions tuned in to a special edition of *CBS Reports* which focussed on the controversy surrounding *Silent Spring*.⁶⁴¹ Carson's calm, thoughtful and dignified demeanour on the television programme belied the industry's depiction of her and, although the programme was intended to be an unbiased account of the debate, the reaction from viewers indicated that the majority had been convinced by her arguments.⁶⁴² Two weeks later the public response resulted in political interest, as Carson was invited to testify to a United States Senate subcommittee on pesticides in 1963. Following Carson's testimony at the Senate hearings, a number of senators commented that her book would 'change the course of history'.⁶⁴³

⁶³⁹ Lear, *Witness to Nature*, 430.

⁶⁴⁰ *Ibid.*, 428-37.

⁶⁴¹ *Ibid.*, 447-52.

⁶⁴² This is despite that some of Carson's colleagues, including nutrition writer Beatrice Trum Hunter, 'found the program to be unbalanced in many ways. Although purportedly the program was an attempt to give equal opportunity to an expression of opposing viewpoints, it was weighted heavily, even if inconsistently, in favor of the chemical approach'. Hunter was a key influence on Carson's environmental research. A series of letters between Carson and Hunter during the late 1950s and up until Carson's death, including an eight-page litany of resources listing the dangers of DDT which Hunter wrote to Carson in 1958, indicate how Hunter helped convince Carson to embark on what would become *Silent Spring*. The letters can be found in the Beatrice Trum Hunter Collection, Howard Gotlieb Archival Research Center at Boston University. Letter from Beatrice Trum Hunter to Rachel Carson, 4 April 1963, Beatrice Trum Hunter Collection, Box 23.

⁶⁴³ Lear, *Witness to Nature*, 448-454.

It is difficult to say, however, if the senators' prediction came true. While *Silent Spring* is largely acknowledged to have marked the emergence of the modern environmental movement, in some ways its legacy has been more of polarising opinion on the topic of environmental stewardship and the safety of the food supply, rather than unifying sentiment around such issues.⁶⁴⁴ Moreover, the controversy surrounding the Delaney Clause and *Silent Spring*, along with governmental reluctance to toughen or even enforce existing food additive legislation, suggested to certain Americans that corporations and the government were simply not to be trusted when it came to the question of food safety. While other contemporary regulatory crises, most notably the thalidomide scare, contributed to such beliefs, the rebellious, anti-authoritarian climate of the 1960s and early 1970s, fostered by the civil rights movement, student unrest, drug culture, Vietnam War protests and the Watergate scandal, also engendered a general spirit of distrust amongst a growing number of Americans.⁶⁴⁵ One of the many ways Americans expressed such dissent was by changing their diet to an organic or natural regime, free of additives, and untainted by industry. As defenders of the food industry Elizabeth Whelan and Frederick Stare remarked in *Panic in the Pantry*, 'the "us versus them" attitude expressed ... is an example of a broad feeling of suspicion the "consumer-

⁶⁴⁴ These divisions were reflected, for example, in the comments made by the participants in a Wellcome Witness seminar on the fortieth anniversary of *Silent Spring* in 2002. D. Christie and E. Tansey (eds.), *Environmental Toxicology: The Legacy of Silent Spring*, Wellcome Witnesses to Twentieth Century Medicine, 19 (2004), Wellcome Trust Centre for the History of Medicine at UCL, London, www.ucl.ac.uk/silva/histmed/downloads/c20th_group/wit19 accessed 23 July 2008.

⁶⁴⁵ Thalidomide, developed by German pharmaceutical company Grünenthal, had been prescribed to pregnant women in many countries, including Germany, Britain and Canada, to combat morning sickness. Tragically, the drug was teratogenic, and caused the infants these mothers gave birth to suffer from severe, often fatal, birth defects, amounting to 10 000 cases during the late 1950s and early 1960s. In the United States thalidomide did not reach the market because of the stubborn questioning of a FDA medical officer, France Oldham Kelsey (b. 1914), who suspected the drug's safety. President Kennedy awarded Kelsey the President's Award for Distinguished Federal Civilian Service for her efforts in stalling the drug's approval. Despite the fact that tragedy had been averted, the manner in which the story broke in the American media, most notably by Morton Mintz in the *Washington Post*, highlighted the inadequacies of state regulators such as the FDA, and suggested that, had it not been for Kelsey, another 10 000 babies would have been deformed or killed in the United States. The controversy aroused not only by the publication of *Silent Spring* in 1962, but also the thalidomide tragedy of the previous year and the passage of the Delaney Clause, spurred concerns not only about the activities of American corporations, but also the ability of the American government to protect the public from harmful or contaminated products. Morton Mintz, *The Therapeutic Nightmare* (Boston: Houghton Mifflin, 1965), 248-64; Rock Brynner and Trent D. Stephens, *Dark Remedy: The Impact of Thalidomide and its Revival as a Vital Medicine* (Cambridge, MA: Perseus, 2001), 39-59.

environmentalists” have for the “greedy industrialists”. It all started with the publication of Rachel Carson’s *Silent Spring*.⁶⁴⁶

Organic Food: ‘It’s no longer a fad, it’s a movement’

Although Whelan and Stare proceeded to lambast the ‘food faddists’ who trumpeted the necessity of a natural diet during the 1960s and 1970s, journalists such as Sandra Blakeslee observed that ‘more and more shoppers are beginning to eye the labels on products with suspicion, trying to find out whether the foods they shake, brown, heat and whip are really safe to eat’.⁶⁴⁷ In turn, the proliferation of natural food cookbooks, health food stores and organic restaurants during these years suggested that a significant segment of the American population were searching for natural, additive-free foods. For example, in 1972 Alice Waters opened the groundbreaking restaurant Chez Panisse, which emphasised its use of local, seasonal and unprocessed ingredients. While counter-culture publications such as *Rat* and *Good Times* published numerous articles on the political and health benefits of an organic diet, natural food was also discussed in the major newspapers and on television in nationally broadcasted programmes such as the ABC and CBS Evening News.⁶⁴⁸ In a general sense, as journalist Jacquin Sanders described in a 1970 newspaper article:

there is a growing repugnance to the things people do to the things people eat. As a result, more and more people are turning to organic food – produce that contains no trace of the chemicals, hormones, antibiotics, preservatives and dyes which have changed the appearance and taste of practically everything that goes into the human stomach. ... It’s no longer a fad, it’s a movement. ... Says Louis Martucci, owner of an organic food store in San Francisco: ‘When we started 12 years ago, our customers were elderly people. High school kids

⁶⁴⁶ Whelan and Stare, *Panic in the Pantry*, 47.

⁶⁴⁷ Sandra Blakeslee, ‘Food Safety a Worry in Era of Additives’, *New York Times*, 9 November 1969, 1, 74, at p. 1.

⁶⁴⁸ ABC Evening News, 15 December 1970, www.tvnews.vanderbilt.edu/diglib-fulldisplay.pl?SID=20080613905212402&code=tvn&RC=8753&Row=18, accessed 13 June 2008; CBS Evening News, 8 November 1970, www.tvnews.vanderbilt.edu/diglib-fulldisplay.pl?SID=20080613905212402&code=tvn&RC=207475&Row=19, accessed 13 June 2008; Warren Belasco, *Appetite for Change: How the Counterculture Took on the Food Industry* (Ithaca, NY: Cornell, [1989] 2007), 29-30.

used to open the door and laugh at us. Now the same type of kid is our main customer.⁶⁴⁹

Martucci's comment that his previous customers tended to be older reflected a previous trend in whole food diets, in which vitamin supplements often played a key role in a healthy diet. As historian Catherine Carstairs has suggested, the whole food advocates of the previous generation, particularly Gaylord Hauser (1895-1984) and J. I. Rodale (1898-1971), but also Adelle Davis (1904-1974), focussed on sales to middle-aged or elderly Americans, and emphasised that their dietary regimes promoted longevity.⁶⁵⁰ Hauser's best-selling book *Look Younger, Live Longer* (1950) and Rodale's publication *Prevention* magazine, founded in the same year, were indicative of this trend.⁶⁵¹

The desire for longevity and disease prevention continued to encourage Americans to pursue an organic diet during the 1960s and 1970s, but as shop owner Louis Martucci suggested, young people were increasingly interested as well. Some health food advocates, such as Maryland store owner Oliver Popenoe, recognised the division between newer organic food stores which emphasised 'the idea of living in harmony with nature, rather than trying to conquer nature' and the previous type of "'pill stores" which tend to be for the old folks with a kind of faintly medicinal atmosphere'.⁶⁵² Or, as Rodale stated, 'only a few years ago the organic health movement was an old people's crusade. Visitors to our farm were almost

⁶⁴⁹ Sanders, 'Organic Food', 103.

⁶⁵⁰ Catherine Carstairs, presentation to the Canadian Society for the History of Medicine, Vancouver, British Columbia, 1 June 2008. Although Hauser lived to the age of 89, Rodale died rather infamously of a heart attack while he was a guest on the Dick Cavett show. Minutes before he had been bragging that he had 'decided to live to a hundred' and that he 'had never felt better'. Dick Cavett, 'When that Guy Died on My Show', *New York Times*, 3 May 2007, www.cavett.blogs.nytimes.com/2007/05/03/when-that-guy-died-on-my-show/, accessed 12 June 2008.

⁶⁵¹ Many other contemporary dieting gurus, such as the low-carbohydrate advocate Robert Atkins (1930-2003), concentrated chiefly on providing regimens for weight-loss.

⁶⁵² Jeannette Smyth, 'Stores Do a Healthy Business in Natural Food', *Washington Post*, 28 February 1971, 129, G4, at p. 129.

always white-haired men and women ... close to the day of reckoning who wanted to stretch life a few more years.⁶⁵³

The dichotomy of old and new health food stores notwithstanding, people chose organic diets for a variety of reasons. Organic food, for example, was a political issue for many advocates. Sharon Grant, the spokeswoman for Mother Nature on the Run Caterers, a co-operative catering company, explained in 1972 that her choice for employment was the culmination of a political transformation which spanned numerous social issues:

I started getting into movements. I got involved with the political campaign of a black guy in New Haven – that’s where I got radicalized – then with a businessmen’s peace group here in Washington ... I took off with a friend and went camping in Canada and Vermont. That really affected me. It was the first time I hadn’t worked and it opened my eyes, showed me what was good for me. Right then I knew that I wanted my pace of life to slow down.⁶⁵⁴

Natural food was also associated with consumer advocates, globally-minded socialists, counter-culture radicals and whistleblowers, as books such as Beatrice Trum Hunter’s *Consumer Beware! Your Food and What’s Been Done to It* (1970), Frances Moore Lappé’s *Diet for a Small Planet* (1971), Ita Jones’ *The Grubbag: An Underground Cookbook* (1971), and former FDA scientist Jacqueline Verrett’s *Eating May Be Hazardous to Your Health* (1974) suggested.⁶⁵⁵

It is important to emphasise, however, that many different political views and approaches were reflected in the organic food movement. Some, such as consumer advocate and Harvard-trained lawyer Ralph Nader (b. 1934), were willing to work within the political system and lobby Congress to force the manufacturers of hot dogs and baby food, for

⁶⁵³ Wade Greene, ‘Guru of the Organic Food Cult’, *New York Times*, 6 June 1971, SM30, SM54, SM56, SM58, SM60, SM65, SM68, SM70, at p. SM65.

⁶⁵⁴ Anonymous, ‘Catered Health Foods, for Washingtonians, “More Curious than Committed”’, *New York Times*, 6 March 1972, 38.

⁶⁵⁵ Levenstein, *Paradox of Plenty*, 179. For more on the link between American counter-culture and diet, see: Belasco, *Appetite for Change*.

example, to reduce the amount of additives included in their production.⁶⁵⁶ Others, such as ‘Marcia’, the columnist for *Good Earth*, believed that dietary change was ‘part of our total revolution’. Writers for *Good Times* and *Rat* added that not only was processed food adulterated in order to ensure ‘a drugged, poisoned, sick, mentally deranged populace’, but also the ‘vested interests of the U.S. are far too strong for them to revalue their approach’.⁶⁵⁷

Other critics of processed food adopted different perspectives, however. For Beatrice Trum Hunter (b. 1918), whose first foray into writing natural food books was *The Natural Foods Cookbook* (1961), natural food was certainly about health, but it was also a matter of environmental responsibility and consumer activism.⁶⁵⁸ Hunter was strongly influenced by Arthur Kallet and F. J. Schlink’s *100,000,000 Guinea Pigs* (1933), a best-selling indictment of the American food, drug and cosmetics industry, and the government agencies and legislation expected to protect American consumers.⁶⁵⁹ Kallet and Schlink urged consumers not only to be vigilant regarding the products they purchased from the supermarket and pharmacy, but also to lobby government agencies, legislators and newspapers about ‘the uncontrolled adulteration and misrepresentation of foods, drugs, and cosmetics’.⁶⁶⁰

Similarly, Hunter criticised the FDA for its inability to protect the safety of the food supply, particularly with respect to food additives. She believed that ‘the FDA has relinquished its mandated control of food additive safety testing to the very industries it was supposed to regulate. The effects are significant insofar as such policies may affect the well-being and very lives of the entire population.’⁶⁶¹ Such criticisms of both industry and government with

⁶⁵⁶ Levenstein, *Paradox of Plenty*, 170-1.

⁶⁵⁷ Belasco, *Appetite for Change*, 32-3.

⁶⁵⁸ Beatrice Trum Hunter, *The Natural Foods Cookbook* (New York: Pyramid, 1961).

⁶⁵⁹ Written correspondence with Beatrice Trum Hunter, 2 March 2007.

⁶⁶⁰ Arthur Kallet and F. J. Schlink, *100,000,000 Guinea Pigs: Dangers in Everyday Foods, Drugs, and Cosmetics* (New York: The Vanguard Press, 1933), 296-303.

⁶⁶¹ Hunter, *The Mirage of Safety*, 8.

regard to food safety were similar to those made by other contemporary consumer activists, such as Ralph Nader, who also pushed for tougher regulations in the auto and nuclear power industries. Although Hunter was influenced by the philosophy of the clinical ecology movement, and Theron Randolph in particular, the tone of her books, as well as her role as Food Editor of *Consumer Research Magazine*, indicated how improving consumer protection through better regulation was a central aspect of her thinking with regards to food. While they were waiting for government regulations to improve, Hunter advocated that consumers educate themselves about food additives and shop selectively. Only half jokingly, she suggested in a *New York Times* interview that, in order to avoid food additives, people should ‘shop around the walls of the supermarkets, where you find real food. All the fake food is in the middle aisles.’⁶⁶²

In the case of Frances Moore Lappé (b. 1944), dietary choice was more than a matter of individual health and consumer rights; it was a global political statement. In her chapter, ‘Recipe for a Personal Revolution’, Lappé insisted that ‘what we eat is within our control, yet the act ties us to the economic, political, and ecological order of our whole planet. Even an apparently small change— consciously choosing a diet that is good for both our bodies and the earth— can lead to a series of choices that transform our whole lives.’⁶⁶³ Indeed, Lappé recalled feeling appalled when, following what she believed to be a ‘rousing political speech’ in 1972, she was asked a question about ‘the difference between long grain and short grain brown rice’. Shocked by such a banal question, she:

wilted. I had wanted to convey the felt-sense of how our diet relates each of us to the broadest questions of our food supply for all of humanity. I had wanted

⁶⁶² Beatrice Trum Hunter quoted in Patricia Wells, ‘An Ire Fed by Fabricated Foods’, *New York Times*, 24 June 1978, 12. Although Hunter gave this advice ‘with a laugh’, most North American supermarkets were designed to have fresh fruit and vegetables, meat and fish, dairy products and bread around the edge of the store, while packaged and frozen foods were aligned in rows in the middle. Other, more conservative nutritionists also advised shoppers to concentrate ‘around the walls’. Thanks to Rima Apple for advising me about this.

⁶⁶³ Frances Moore Lappé, *Diet for a Small Planet* (New York Ballantine, [1971] 1991), 8.

to convey the way in which economic factors rather than natural agricultural ones have determined land and food use. Was I doing just the opposite? Was I helping people to close in on themselves, on their own bodies' needs, instead of using the information to help them relate to global needs?⁶⁶⁴

Despite Lappé's concerns, however, many people continued to choose an organic diet for personal, rather than political, reasons. According to journalist Jeannette Smyth, there were those who believed that a natural diet could be spiritually, as well as physically, beneficial, and fit into a lifestyle that might also include yoga and transcendental meditation.⁶⁶⁵ For others diet was the hub around which their desire to return to a more natural, communal form of life was centred. As historian Warren Belasco has described, those Americans who were interested in returning to an era where particularly vilified additives, such as those derived from petrochemicals, were absent observed two maxims: 'don't eat anything you can't pronounce ... and if worms, yeast and bacteria grew on it, then it must be natural, for no self-respecting bug would eat plastic'.⁶⁶⁶

Others groups that turned to organic foods included the increasing numbers of people who were sensitive to the additives found in food, people treated by physicians such as Theron Randolph. Mysterious conditions such as 'Chinese Restaurant Syndrome', attributed by Chinese-American physician Ho Man Kwok to monosodium glutamate (MSG) in a 1968 letter to the *New England Journal of Medicine*, reinforced the idea put forth by Randolph that food additives could cause chronic health conditions and that certain people were particularly

⁶⁶⁴ Lappé, *Diet for a Small Planet*, 3.

⁶⁶⁵ Smyth, 'Stores do a Healthy Business', G4.

⁶⁶⁶ Belasco, *Appetite for Change*, 40.

sensitive.⁶⁶⁷ Finally, there were those such as English instructor Sylvia Feldman who believed simply that organic food ‘tastes better. ... That’s why I started buying organic.’⁶⁶⁸

The profitability of organic food during the late 1960s and early 1970s meant that many of those who owned health food stores were not primarily spurred by politics. For many shop owners and grocery store chains, organic food was simply a prosperous enterprise, one that was attracting the attention of Wall Street investors.⁶⁶⁹ As journalist Jean Hewitt noted in 1971:

Whether fad or trend, health food stores are multiplying in both city and suburb. Some of the shops are individually owned and operated, but more and more they are members of chains that must see financial growth through the increase in ecology-minded consumers who have turned to natural and organic foods. The little dusty health food store with a limited line of products is becoming extinct; the new stores, well-stocked and in busy locations, obviously aren’t depending simply on trade from hippie-types who led the crusade for organic foods.⁶⁷⁰

Organic grocer and non-organic restaurateur, Lester Grossman concurred, admitting in the same year that ‘I suppose the reason I am this involved is that it’s a very good industry to be in. ... Organic food is on the upswing ... where most other industries in the country are on the downswing’.⁶⁷¹ Another health food store owner added: ‘Look ... I’m a merchant, not a missionary. We have no other causes— no political causes, no nothing. I just supply nice,

⁶⁶⁷ Kwok described that the most prominent symptoms were ‘numbness at the back of the neck, gradually radiating to both arms and the back, general weakness and palpitations’. Ho Man Kwok, ‘Chinese Restaurant Syndrome’, *New England Journal of Medicine* 278 (1968), 796. Historian Ian Mosby has argued that the ‘many of the basic assumptions about the Chinese restaurant syndrome were, at core, the product of a racialized discourse that framed much of the scientific, medical and popular discussion surrounding the condition’, giving medical legitimacy to ‘the strangely “exotic”, “bizarre” and “excessive” practices associated with Chinese culture’. This reflected the views of one of the interviewees for this thesis, but others believed that ‘ethnic’ food, particularly, Italian, Kosher and Asian cuisine, was less likely to contain additives. Shula Edelkind, Interview; Paula Kimball, Telephone Interview, 4 February 2008; Anonymous, Telephone Interview, 4 February 2008; Anonymous, Email Interview, 20 May 2008; Ian Mosby, ‘“That Won-Ton Soup Headache”: The Chinese Restaurant Syndrome, MSG and the Making of American Food, 1968-1980’, *Social History of Medicine* 22 (2009), 133-51, at p. 134.

⁶⁶⁸ Jean Hewitt, ‘Organic Food Fanciers Go to Great Lengths for the Real Thing’, *New York Times*, 7 September 1970, 23.

⁶⁶⁹ Robert A. Wright ‘Health Foods – Only a Fad?’, *New York Times*, 15 October 1972, F1, F5.

⁶⁷⁰ Jean Hewitt, ‘Buying Health Foods is Easier Now’, *New York Times*, 19 June 1971, 14.

⁶⁷¹ Smyth, ‘Stores do a Healthy Business’, G4.

good food.⁶⁷² Nevertheless, other store owners felt somewhat guilty taking advantage of people who appeared to be merely jumping on the bandwagon of organic food. Although James Kennedy started Kennedy's Natural Foods because he and others 'super-sensitive' to additives were tired of 'foraging the countryside for food', he recognised how stories in the media about the dangers in a particular processed food resulted in much higher sales for its natural alternative.⁶⁷³

Concern about food additives and interest in organic foods also entered the political arena during the early 1970s, as a series of governmental hearings weighed into the issue.

Connecticut's Senator Abraham Ribicoff (1910-1998), for example, expressed his concerns about food additives in his opening address as chair of the 'Chemicals and the Future of Man' hearings (1971):

It is a common saying that we are what we eat. If this is true, then Americans are becoming a nation of processed, packaged, and preserved people. Last year, Americans bought more processed than fresh foods for the first time in our history. We spent more than \$60 billion for these convenience foods including such items as TV dinners, snack foods of all kinds, and frozen foods. With these foods we each consume every year more than four pounds of chemical preservatives, stabilizers, colorings, flavorings, and other additives. ... Today more than 3,000 chemicals are deliberately added to our foods. These developments raise three basic questions: (1) How much do we know about the hazards to human health from these chemicals? (2) How much assurance of chemical safety should we require? (3) What must the federal government do to assure that the chemicals we absorb are safe?⁶⁷⁴

Another Senator, Gaylord Nelson of Wisconsin (1916-2005), introduced legislation to ban untested additives by stating:

People are finally waking up to the fact that the average American daily diet is substantially adulterated with unnecessary and poisonous chemicals and frequently filled with neutral, nonnutritious substances. We are being chemically medicated against our will and cheated of food value by low nutrition foods. It is time to take a careful look at the prolific use of additives permeating our foods. ... The profits of the food industry are being placed above the public health as regards the safety, nutrition, and necessity of food

⁶⁷² Ibid.

⁶⁷³ Ibid.

⁶⁷⁴ Abraham Ribicoff quoted in Hunter, *Mirage of Safety*, 1.

additives. Synthetic and convenience foods mean high profits and greater market control of the food industry.⁶⁷⁵

Other Senate and Congressional hearings were held during the early 1970s, including those on particular food additives, such as the cyclamates used as artificial sweeteners, the synthetic hormone diethylstilbestrol (DES) and nitrites and nitrates.⁶⁷⁶ There were other contemporary hearings on more general aspects of food additives and nutrition, often chaired by prominent politicians such as Senator Edward Kennedy (b. 1932) and Democratic presidential nominee George McGovern (b. 1922).

Not surprisingly, Kennedy and McGovern would become involved in the debate about the Feingold diet. Kennedy was unimpressed by the FDA's ability to take firm action with regards to Feingold's theory. His frustration with the FDA was expressed at a meeting of the Senate Labor and Public Welfare Committee's Subcommittee on Health. According to the *Los Angeles Times*, Kennedy was:

anxious for a study to begin. 'We want to run this thing out and test it,' he said. 'If you're not doing it, we want to know why. If it's because of a lack of funds, we want to fulfill our legislative responsibility.' Dr. Albert Kolbye, associate director for science of the FDA's bureau of foods, said, 'We're getting ourselves together, Senator.' 'You'll have to do better than that,' Kennedy said. 'We will not tolerate a mish-mash of government agencies which all want a piece of the action,' Kennedy said, 'The commissioner of the FDA must indicate that this is a priority action.'⁶⁷⁷

Although Kennedy would be disappointed by the FDA's response to Feingold's hypothesis, the FDA did ban some substances, most notably cyclamates and DES.⁶⁷⁸ Nevertheless,

⁶⁷⁵ Gaylord Nelson quoted in Hunter, *Mirage of Safety*, 2, 5.

⁶⁷⁶ Hunter, *Mirage of Safety*, 304-5.

⁶⁷⁷ Marlene Cimon, 'Hyperactivity and Food Additives', *Los Angeles Times*, 15 September, 1975, D1, D6, D8, at p. D6.

⁶⁷⁸ According to Sheldon Krimsky, evidence that the synthetic hormone DES was carcinogenic was one of three key factors that led to the development of the 'environmental endocrine hypothesis', a theory which posits 'that a diverse group of industrial and agricultural chemicals in contact with humans and wildlife have the capacity to mimic or obstruct hormone function – not simply disrupting the endocrine system like foreign matter in watchworks, but fooling it into accepting new instructions that distort the normal development of the organism'. The other two factors included the emergence of a large number wildlife studies linking reproductive disorders to industrial and agricultural effluents and research postulating 'global decline in the

critics, such as Beatrice Trum Hunter, continued to charge that the FDA and the USDA were too lenient in allowing harmful additives into the marketplace.⁶⁷⁹ Indeed, critics bemoaned the fact that the FDA investigated individual chemicals only on a case by case basis – and only after a great deal of public outcry – rather than considering the safety of broad categories of additives, such as those which were essentially petrochemical products.⁶⁸⁰ In contrast, many nutrition scientists, most notably Harvard's Frederick Stare, argued that the regulators had gone too far and that this was 'making eating a less enjoyable experience'.⁶⁸¹ With regards to what he and co-author Elizabeth Whalen called 'cyclamania', Stare argued that, because of the Delaney Clause, 'a cancerphobic American public was willing to ban a substance upon hearing the merest shred of evidence'.⁶⁸² The authors proceeded to state that 'if the Delaney clause had not been exerting its force, it is unlikely that the general public anxiety about food additives would be as intense as it is now. Healthfoodland would be a remote hideaway for eccentric people, instead of the billion-dollar business it is today.'⁶⁸³

Stare's implication that health food advocates were more concerned with profit than health was disingenuous given his own connections to the food industry and the reliance of the Harvard Department of Nutrition, which he founded in 1942 and continued to chair until

quality and quantity of human sperm'. Krinsky's analysis of the social factors that contributed to the development and promotion of this hypothesis bears some parallels to the popularisation of Feingold's hypothesis. Krinsky, *Hormonal Chaos*, 2-4.

⁶⁷⁹ Ironically, by banning cyclamates, the FDA was accused of bowing to the pressure of the sugar industry lobby. Anonymous, 'We've Been Asked How Healthful is "Health Food?"' *U.S. News and World Report*, 21 July 1975, 64; Hunter, *Mirage of Safety*, 198-225.

⁶⁸⁰ Belasco, *Appetite for Change*, 140.

⁶⁸¹ Whalen and Stare, *Panic in the Pantry*, 6.

⁶⁸² *Ibid.*, 154-60. According to an anonymous editorial in the *Lancet*, the evidence used to demonstrate that cyclamate was a carcinogen was insufficient. Only four of the fifty rats developed bladder cancer and, since the researchers had fed the rats a mixture of saccharin and cyclamate, it was difficult to determine exactly what caused the carcinoma. The author asked, 'was the fate of the world's sweet tooth decided just on the response of ... 4 rats?' Ultimately, the editorial blamed the Delaney Clause for over-simplifying how scientists determined what and how particular substances were carcinogenic. In the case of food additives and cancer, as with food additives and hyperactivity a few years later, some physicians were unwilling to denounce substances before 'a detailed understanding of the mechanisms involved' was determined. Anonymous, 'Why Cyclamates Were Banned', *Lancet*, 295 (1970), 1091-92.

⁶⁸³ Whalen and Stare, *Panic in the Pantry*, 163.

1976, on corporate funders.⁶⁸⁴ Other prominent supporters of the food industry, such as Stare's Harvard colleague, Jean Mayer (1920-1993), also had connections to the food, drug and chemical industries, serving on the Boards of Directors of Monsanto and Miles Laboratories.⁶⁸⁵ On the other hand, Stare was correct in observing that health food had become big business by the 1970s, and the comments of organic food store owners attested to this development. Another example was J. I. Rodale's health-food press, which made over nine million dollars in 1970.⁶⁸⁶ Even as the organic food movement faded during the late 1970s and 1980s, food companies were nevertheless able to develop products which capitalised on the desire of 'yuppies' for 'health' food.⁶⁸⁷ The role of economics, as well as politics, on both sides of the debate over food additives, made it difficult for consumers to delineate if there was a boundary between ideology and nutrition science.

For parents, especially those whose politics, environmental concerns and health had not conditioned them to favour one side of the debate, it was difficult to weigh the opinions of various scientific authorities regarding the dangers of food additives and the benefits of organic food. Both sides of the debate over food additives were typically represented in the media, providing no easy answers. Even the safety of the humble maraschino cherry was difficult to determine. While the World Health Organisation deemed the red dye used in the cherries to be carcinogenic, the FDA believed that the amount of dye used was too minimal to warrant a ban.⁶⁸⁸ The familiarity of products such as maraschino cherries made it difficult for

⁶⁸⁴ D. Mark Hegsted, 'Frederick John Stare (1910-2002)', *Journal of Nutrition* 134 (2004), 1007-9; Anonymous, 'Frederick Stare', *The Economist*, 18 April 2002, www.economist.com/obituary/displaystory.cfm?story_id=E1_TDRGGRI, accessed 17 June 2008.

⁶⁸⁵ Belasco, *Appetite for Change*, 114. Mayer was less comfortable with the connections between nutrition science and the food industry, and some have argued that this was one reason why he left Harvard to become president of Tufts University in 1976. Mayer's attitude about the Feingold diet was similarly ambiguous, as is demonstrated in Chapter 8. With regards to pesticides, Mayer recommended a fairly balanced approach, stating that the 'big problem has been the indiscriminate use of pesticides in the past' but also that there 'is no sense in going from one extreme to the other and going back to the Cave Age'. Jean Mayer quoted in Jean Hewitt, 'Organic Food Fanciers', 23; Anonymous, 'In Person Interview', 8 December, 2007.

⁶⁸⁶ Greene, 'Guru of the Organic Food Cult', SM54.

⁶⁸⁷ Belasco, *Appetite for Change*, 1-3.

⁶⁸⁸ Sandra Blakeslee, 'Challenge to Food Tests', *New York Times*, 10 November 1969, 1, 51, at p. 1.

some families to believe that typically American foods were dangerous. As journalist Elaine Jarvik described in an article featuring two Utah families on the Feingold diet:

Even at a time when Americans had begun to wonder if perhaps they were, indeed, what they ate, Dr. Feingold's diet was not so easy to swallow. Here was a man warning of evils lurking in something as innocuous as frozen french fries, as healthy as toothpaste, as all-American as hot dogs. It sounded, to some, a little paranoid, and about as plausible as grapes and copper bracelets for cancer and arthritis.⁶⁸⁹

Different messages also emerged from the medical community. On the one hand, the AMA stated in 1969 that 'there is no reason to believe that the present use of chemicals in foods is endangering the health of the people'.⁶⁹⁰ In addition, George Christakis of the Mount Sinai Medical Center in New York described organic foods as a 'public health threat' due to the false health claims made about them.⁶⁹¹ In contrast, an editorial in the *Lancet* warned that 'the question of the ultimate effects of food additives on man is unanswered. Human experiments are possible only on a very small scale, and, in any case, they do not mimic the life-long, very low doses to which man is exposed.'⁶⁹² Nobel laureate and geneticist Joshua Lederberg (1925-2008) agreed with the *Lancet* editorial, stating that 'it would be held a catastrophe if only a hundred U.S. consumers a year were carcinogized by a food additive they could happily live without'.⁶⁹³

Similarly, parents who would employ the Feingold diet, as with other Americans, came to the notion that food additives could be harmful from a number of different perspectives. While some were already convinced of the benefits of an organic, natural diet, others were sceptical that food additives could be the cause of their child's hyperactivity. Lora Hollins, for

⁶⁸⁹ Elaine Jarvik, 'The Calming of the Hyperactive', *Utah Holiday*, May 1978, 48-50, 63, at p. 48.

⁶⁹⁰ AMA quoted in Sandra Blakeslee, 'Challenge to Food Tests', 1, 51.

⁶⁹¹ George Christakis quoted in Grace Lichtenstein, "'Organic' Food Study Finds Pesticides', *New York Times*, 2 December 1972, 39.

⁶⁹² Anonymous quoted in Blakeslee, 'Challenge to Food Tests', 51.

⁶⁹³ Joshua Lederberg quoted in Harrison Wellford and Samuel Epstein, 'The Conflict over the Delaney Clause' *New York Times*, 13 January 1973, 31.

example, described herself as a ‘health food freak’ who had been influenced in her late teens by Adelle Davis’ books on nutrition. Believing that many of her allergies were due to reactions to food dyes, she avoided them to the point that the only food in her house that was ‘artificially coloured or flavoured was margarine, pancake syrup and occasionally maybe some ice creams that were coloured’. Although the dyes in these products caused problems for her son, she found out that most of the additives he was consuming were provided in foods he ate at school.⁶⁹⁴ Similarly, an anonymous parent ‘had always been interested in nutrition’ and baked her own bread. Her son rarely came into contact with food additives, but nevertheless reacted strongly to many naturally occurring salicylates, particularly in grapes and tomatoes.⁶⁹⁵

In contrast, other parents had little apprehension about food additives before hearing about the Feingold diet. For instance, Shula Edelkind lived in Nigeria for a year with her children and, since all she could get was powdered milk, she ‘used to put red food colouring in the milk and sugar because it didn’t taste so great’.⁶⁹⁶ Other parents, such as Texan Marilee Rigg, cooked most meals from scratch, but had not been in the habit of reading labels, and thought nothing of giving their children drinks, desserts and chewing gum containing artificial colours and flavours.⁶⁹⁷ Although Susan Leitner refused to give her son Ritalin, she recalled being ambivalent about food additives. Once she began using her skills as a librarian to research the Feingold diet, however, she discovered literature which warned about food additives and decided to try the diet.⁶⁹⁸

Conclusion

⁶⁹⁴ Lora Hollins, Interview.

⁶⁹⁵ Anonymous, Telephone Interview, 5 February 2008.

⁶⁹⁶ Shula Edelkind, Interview.

⁶⁹⁷ Marilee Rigg, Telephone Interview, 20 May 2008.

⁶⁹⁸ Susan Leitner, Interview.

During the post-war period, the amount of food additives found in the American diet increased markedly. Almost immediately, as the 1950-1951 Delaney hearings indicated, they became a source of controversy, although it took until the late 1960s for the furore over food additives to become front page news. Such developments occurred partly because of the Delaney Clause and Rachel Carson's *Silent Spring*, but also because of an emerging culture of mistrust within the United States. By the early 1970s, the debate over food additives also highlighted political schisms within the nation, and differentiated between those who had faith in the food industry and federal regulatory agencies and those who did not. Trusting the government over whether food additives were safe was not so different than believing its claims about the war in Vietnam, about the threat posed by drugs such as marijuana and LSD, or about the fidelity of the American president. It was into this divisive context that the Feingold diet emerged. Given the cultural climate of suspicion during the early 1970s, it is not surprising that the media found Feingold's claims so captivating, and that the food industry found them so alarming.

Food additives, however, were not simply a matter of politics. Although critics such as Whalen and Stare tended to lump all of those who warned about food additives, including Feingold, into a homogeneous conglomeration, those concerned about food additives came to the issue from many different perspectives.⁶⁹⁹ As highlighted by journalist Wade Greene in 1971, the organic food movement included:

a wide variety of food cultists, from old-line vegetarians to youthful Orient-oriented 'macrobiotic' dieters ... plus reactionaries yearning to turn back all clocks, urban dropouts in search of simpler, more natural lifestyles, ecologists who are worried about the long-range environmental effects of some chemicals, Dr. Strangelove paranoids who read poison plots on the ingredient labels of pancake mixes and, increasingly, rather ordinary folk to whom pronouncements about the perils of cyclamates, DDT, mercury, monosodium

⁶⁹⁹ Whalen and Stare, *Panic in the Pantry*, 114-15.

glutamate, phosphates, etc., have stirred a wariness about all man-made chemicals, particularly those that get in their food.⁷⁰⁰

Food additives may have been despised equally by the counterculture anarchist, the environmental toxicologist and the food allergy sufferer, but for quite different reasons. What the anarchist may have interpreted as a plot machinated by a corrupt government and greedy industrialists, the toxicologist may have seen as another indication of western society's perilous, but perhaps unwitting, descent into an increasingly polluted environment. In contrast, food allergy sufferers might have perceived food additives on a much more intimate level, viewing them as simply another barrier they faced to a life free of chronic illness.

In other words, the decisions Americans made about food additives were not usually based solely upon the reading of a newspaper article or the watching of a television programme, but were instead a reflection of political beliefs, cultural background, spirituality and, most importantly, personal and familial health experiences. If this is correct, then it highlights one of the challenges inherent in any sort of preventative health policy: people are resistant to break habits for the benefit of their health unless personal experiences dictate that they do so. As numerous historians of nutrition and food have outlined, it has been difficult for physicians and policy makers to convince people to change their diet, even if such changes were said to be salubrious or economical, partly because 'everyone thinks that they are an expert on their own diet'.⁷⁰¹ But when the development of an ideology and/or an experience of ill health provides the necessary evidence for an individual that breaking a habit, whether it be smoking, drinking alcohol or consuming food additives, is warranted, then such beliefs can belie the sanctions of even the highest medical authority. The next section takes up such issues by evaluating how the Feingold diet was described and evaluated by the media, physicians and the parents of hyperactive children.

⁷⁰⁰ Greene, 'Guru of the Organic Food Cult', SM30.

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**Into the Mouths of Babes: Hyperactivity, Food Additives and the
History of the Feingold Diet**
[volume 2 of 2 volumes]

Submitted by Mathew Phillip Campbell Smith, to the University of Exeter as a thesis for the degree of DOCTOR OF PHILOSOPHY in HISTORY, June 2009.

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Part III

The Reception of the Feingold Diet

Chapter 8

The Feingold Diet in the Media

Anxiety about food additives persisted into the mid 1970s, in the face of a recession that threatened to undermine consumers' willingness to pay for expensive organic foods. As journalist Anna Colamosca reported in 1974, 'despite soaring prices, the \$600-million health food industry seems to be holding up well'. Although Colamosca stated that 'hundreds of health food stores across the country have gone out of business because they were overcharging in an effort to make a fast buck', she also believed continuing newspaper stories 'related to the food industry have kept many people doggedly returning to their favourite health food stores over the last six months'.⁷⁰² One story she cited was Feingold's recent linkage of food additives and hyperactivity. Just as interest in the Feingold diet was fuelled by concern about food additives, as well as dissatisfaction with treatments for hyperactivity, Feingold's theory also kept food additives in the headlines, while interest in organic foods began to wane and health food sales began to slump.⁷⁰³

The reasons why Feingold embraced the mass media, rather than disseminating his theory through medical publications, were discussed in Chapter 5. It is worth repeating, however, that Feingold did not initially court such attention himself; the AMA was responsible for inviting him to their 1973 and 1974 conferences and organised Feingold's press conferences. Nevertheless, once Feingold decided to reach out to the public with his theory, he did so with an eagerness and energy that belied his age. Ironically, the refusal of the top medical journals to provide Feingold with a forum from which to publish his ideas to his fellow physicians resulted in his idea receiving much greater exposure in the mainstream media; not only were

⁷⁰² Anna Colamosca, 'Health Foods Prosper Despite High Prices', *New York Times*, 17 November 1974, 205.

⁷⁰³ *Ibid.*

parents able to read about the diet in their local newspaper, so too were clinicians more likely to read *New York Times* than *JAMA*.

Feingold's ability to disseminate his ideas through the media was facilitated by increased media interest in health reporting during the post-war period.⁷⁰⁴ Given the intertwining relationships between the media, policy makers, physicians, patients, advertisers and readers, the media's role with regards to health was a complex one. On one level, the reliance of mass media on advertising revenue helps to explain some of these ambiguities. Historians Virginia Berridge and Kelly Loughlin, for example, have described how 'the mass media has been enlisted as a public health tool through the development of mass advertising campaigns, and it has been the focus of opposition and control due to the use of mass advertising by commercial interests such as tobacco and alcohol'.⁷⁰⁵ The role of advertiser, however, was a passive role for the media compared to its role as a purveyor and interpreter of news and a teller of stories. Both health scares and miracle cures made for compelling stories, ones that generated interest and could have a profound impact on both public policy and the actions of the general public.

Sociologist Clive Seale has emphasised how reports about food scares were particularly apt to attract media interest, stating that 'the depiction of ordinary objects whose ingestion is essential for life, yet nevertheless reveal themselves as threats to life, presents a highly entertaining juxtaposition of opposites for the media health producer'.⁷⁰⁶ The controversial

⁷⁰⁴ Virginia Berridge and Kelly Loughlin, 'Introduction', in Virginia Berridge and Kelly Loughlin (eds.), *Medicine, the Market and the Mass Media: Producing Health in the Twentieth Century* (London: Routledge, 2005), 1-16, at p. 6.

⁷⁰⁵ *Ibid.*

⁷⁰⁶ Health care researchers Martin King and Clare Street have echoed Seale's observations about the popularity of food scare stories. Clive Seale, *Media and Health* (London: Sage, 2002), 70-5; Martin King and Clare Street, 'Mad Cows and Mad Scientists: What Happened to Public Health in the Battle for the Hearts and Minds of the Great British Beef Consumer?' in Martin King and Katherine Watson (eds.), *Representing Health: Discourses of Health and Illness in the Media* (New York: Palgrave Macmillan, 2005), 115-32, at p. 119.

nature of many of the nutrition-related health scares during the post-war period probably added to their appeal. Unlike the thalidomide scandal, however, which was presented as a clear-cut case of corporate greed and incompetence, there were almost always two justifiable perspectives represented in stories involving pesticides, food colours, artificial sweeteners and even the recommended daily allowances of fat, cholesterol and alcohol. The banning of cyclamate sweeteners, for instance, might have been a victory for organic food advocates and the sugar industry, but it dismayed diabetics and nutritionists and physicians concerned about obesity.⁷⁰⁷

Seale's tendency to portray journalists as scaremongers who exaggerated the seriousness of such food crises also overlooks the fact that journalists did not act alone in constructing these stories. In the case of the Aberdeen typhoid outbreak of 1964, for instance, a symbiotic relationship existed between the media and the Medical Officer of Health, Ian MacQueen, with regards to reporting the story of the contaminated corned beef. While MacQueen, who had originally studied journalism, was able to utilise the press to help contain the outbreak and to further his desire to promote health education, the media found the outbreak to be 'a good story', one that was 'intensely reported' and 'began to take on a life of its own'.⁷⁰⁸ Although the Milne Report, an inquiry into the outbreak, criticised the relationship between MacQueen and the media, stating that 'the outbreak and the possible dangers of its spread were exaggerated to such an extent that the incident received publicity out of all proportion of its significance', many Aberdeen physicians supported MacQueen's efforts, and the investigative journalism employed during the outbreak served as a model for later food crises.⁷⁰⁹

⁷⁰⁷ Richard D. Lyons, 'Saccharin Ban Causes Storm of Complaints', *New York Times*, 11 March 1977, A1, A28.

⁷⁰⁸ Lesley Diack and David Smith, 'The Media and the Management of a Food Crisis: Aberdeen's Typhoid Outbreak in 1964' in Berridge and Loughlin, *Medicine, the Market and the Mass Media*, 81-6.

⁷⁰⁹ Diack and Smith, 'Media and Management', 88-9.

With the Aberdeen typhoid outbreak and the BSE crisis of the 1990s the danger posed by tainted beef was unquestionably real. What was questioned in both cases was the media's role in exaggerating the degree of risk and contributing to consumer panic and financial loss in the agricultural and tourism sectors. In the case of more radical or unsubstantiated health claims, the impact of the media can be even greater, especially when mainstream medicine is unwilling to support such claims. In his discussion of the media's role in providing alternative nutrition advice to American consumers, Warren Belasco has argued that 'as health and nutrition counsellors, the media ranked second only to physicians. And since most medical information dealt with weight or acute health problems (like diabetes), the mass media were the principal source of advice and information.'⁷¹⁰ Belasco believes that, with regards to counter-cuisine and the organic food movement, the American media 'sought the high middle ground of conservative reform', rejecting the extreme views of both sides of the debate.⁷¹¹ As this chapter demonstrates, this was not the case with the Feingold diet. Newspapers presented strong opinions on both sides of the debate, often angering both proponents and detractors of the diet.

From the time of his first television appearance on San Francisco television in 1972 and until his death a decade later, Feingold publicised his theory via hundreds of newspaper editorials, magazine articles, radio debates and television programmes. According to Feingold, by 1976 the number of newspaper articles had reached approximately twenty-seven per month, making it, according to a *Los Angeles Times* reporter, 'one of the most widely discussed and

⁷¹⁰ Warren Belasco, *Appetite for Change: How the Counterculture Took on the Food Industry* (Ithaca, NY: Cornell, [1989] 2007), 154-5.

⁷¹¹ *Ibid.*, 155. As sociologist Erin Steuter has observed, the mass media has grown more resistant to alternative medicine during the last fifteen years, and has taken to calling it 'junk science'. Although she does not mention it, it is possible that consolidation of ownership in North American mass media during the same period, particularly by conservative owners (such as Rupert Murdoch) has contributed to this trend. Erin Steuter, 'Peddling Skepticism: Media Representations of Homeopathy as "Junk Science"', *Journal of American and Comparative Cultures* 24 (2001), 1-10.

controversial topics in American medicine'.⁷¹² While the Feingold diet often made the front page, for example in Morton Mintz's story for the *Washington Post* in late October 1973, it was also discussed in sections devoted to food, health, women's issues, parenting and lifestyle.⁷¹³ Feingold's hypothesis even found its way into newspaper quizzes which focussed on current events.⁷¹⁴ The Feingold diet was covered by newspapers read by millions, such as the *New York Times*, and low-circulation magazines such as *Utah Holiday*. Similarly, Feingold was willing to be interviewed not only on nationally syndicated television programmes, such as the *Phil Donahue Show*, the first and longest-running American tabloid talk show, lasting from 1970 to 1996, and NBC's *Today*, hosted by Barbara Walters (b. 1929), but also local programmes. During a visit to Texas in 1978, for example, the Dallas-Fort Worth Feingold Association arranged for Feingold to be interviewed on the local ABC and NBC stations. The NBC interview was conducted by emerging talent, Charlie Rose (b. 1942).⁷¹⁵

Although most media reports of the Feingold diet, especially during the 1970s, tended to be positive, some were negative and others were fairly neutral, or emphasised the controversial nature of the issue. Both positive and negative coverage used arguments about the Feingold diet as a means to achieve political ends that had little to do with helping hyperactive children. As such, discussion of the Feingold diet in the media could become a dispute about the role of government and regulation in a free market system as much as it was a debate about how to explain and treat hyperactivity. As the political climate changed during the 1980s, so too did the tenor of stories about the Feingold diet and the public's willingness to

⁷¹² ; Harry Nelson, 'Hyperactive Children and Diet: Is There a Link?', *Los Angeles Times*, 19 April 1976, B3, B20, at p. B3.

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⁷¹⁴ The 'Saturday News Quiz' asked readers: 'According to recent studies prompted by the popular and controversial "Feingold diet," what substances in food affect the behavior of hyperactive children?'. Linda Amster, 'Saturday News Quiz', *New York Times*, 5 April 1980, 15

⁷¹⁵ Marilou Rigg, Interview.

question clinical approaches to hyperactivity. The Feingold diet existed in the media not only as a captivating story about a charismatic physician and his startling discovery, but also as a barometer for the public's attitude towards the medical community, American corporations and the government's ability to monitor and regulate these entities.

This chapter begins by exploring media stories that were favourable towards the Feingold diet and analysing the motives authors had for supporting Feingold's hypothesis. It then discusses the arguments directed against Feingold in the media. Although most accounts of the Feingold diet were clearly negative or positive, a number were more ambiguous. Among this category of media reports was the 'On Nutrition' column, written by nutrition scientists Jean Mayer, Joanna Dwyer and Jeanne Goldberg. The chapter examines how these authors dealt with Feingold's thesis and contends that, unlike most editorials, 'On Nutrition' reflected the inherent difficulties in evaluating the Feingold diet. It concludes by suggesting why media interest in the Feingold diet faded following the allergist's death in 1982.

‘A precious commodity to hyperactive children?’

The first newspaper articles about the Feingold diet appeared after the allergist presented his findings to the AMA conference in June 1973, and for the next decade, stories about the Feingold diet appeared regularly in American newspapers. Reflecting American concerns about food additives and hyperactivity, newspapers were quick to report on Feingold’s ideas and the controversy that surrounded it.⁷¹⁶ Media coverage of the Feingold diet tended to fall into three primary categories: stories that supported Feingold’s hypothesis; stories that were sceptical or reported on studies which yielded negative results; and relatively neutral stories which represented both sides of the debate. Newspaper and magazine stories could take the form of anonymous reports of trial results, regular columns contributed by a health professional, science reporters or food writers, editorials written by food industry representatives or advocates of the Feingold diet, multi-page feature articles focusing on Feingold families, debates between experts or letters to the editor.

Provocative headlines often made clear the perspectives represented in many stories. For instance, the titles of a pair of stories favourable towards the Feingold diet and published in the *Washington Post* on 23 January, 1975 were ‘Color it Dangerous’ and ‘Coloring Food - Who Suffers?’⁷¹⁷ Negative stories, such as those written in 1977 and 1978 by health columnist G. Timothy Johnson, entitled ‘Food Additive Link to Hyperactivity Unproven’ and ‘Diet-Hyperactivity Link Still Unproved’, also tended to reveal their perspective on the diet in the headline.⁷¹⁸ Similarly, the headlines of neutral stories often focused on the controversial nature of the Feingold diet or posed a question about his theory, such as, ‘Can

⁷¹⁶ One of the newspapers to report on Feingold’s first press conference was apparently so eager to break the story it did not bother to copy-edit their story. Feingold was described in the first sentence as a psychologist, only later to be correctly identified as an allergist, and one of his colleagues, Donald German was incorrectly identified as ‘Daond’ German. Anonymous, ‘Food Additives Tied to Hyperactive Behavior’, 32.

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Dye-Hyped Foods Cause Hyperactivity?’⁷¹⁹ Depending on which story was read, the Feingold diet could be perceived as either ‘A Precious Commodity for Hyperactive Children’ or ‘Another “Miracle” Diet Cure that Failed’.⁷²⁰

Despite the fact that it is possible to categorise stories about the Feingold diet as positive, negative and neutral, these categories existed on a continuum stretching from the extremely positive to the utterly dismissive. While a number of relatively positive stories were simply reports of a successful trial or coverage of a family that had benefited from the Feingold diet, some of the most positive stories were written by ideologically motivated columnists who used Feingold’s theory to berate the government or the food industry. For example, Colman McCarthy’s (b. 1938) glowing review of *Why Your Child is Hyperactive* was representative of the writer’s tendency to criticise the American government and capitalism. As a 1986 article about McCarthy’s dismissal from the faculty of American University described, ‘Mr. McCarthy’s espousals of leftist ideas – on everything from civil disobedience to vegetarianism – rarely fail to ignite a reaction’.⁷²¹ Characteristically McCarthy’s endorsement of Feingold’s book was inflammatory:

Feingold’s book has the ring of alarm to it, as well it should. Such a message is likely to be dismissed as heresy among the true believers who trust the fake food companies and the Food and Drug Administration. Feingold can be quickly put down by those in power: his studies were ‘unscientific,’ they were of limited range, and besides who is he – just a tinkering allergist – to say he has the answers. Doesn’t Feingold know that we must see the bodies falling dead in the street before there is ‘absolute proof’ and action can be taken? ... Too many citizens suspect that they cannot trust the food companies, and they know that the FDA is uncaring or underfunded, or else it would be leading the way to find answers, not telling Feingold to go away merely because he wants the consumer to see clearly that the food he is buying is fake. ... If parents want to act to protect their child, they will likely have to do it on their own. The best help they may get is not from the medical community, the FDA nor the food companies, but from this book.⁷²²

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⁷²⁰ Joan Beck, ‘Another “Miracle” Diet Cure that Failed’ *Chicago Tribune*, July 11, 1977, C2; Monte, ‘Feingold Diet’.

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Not surprisingly, the last two sentences from the quotation above were used in Random House's print advertising campaign for *Why Your Child is Hyperactive*.

An anonymous 1975 editorial published in McCarthy's paper, the *Washington Post*, also criticised the manner in which the FDA was handling Feingold's hypothesis. Reporting on the hearings of a Senate Health Subcommittee, the author urged the FDA to act on the 'unsettling' findings of a study into the Feingold diet, one led by C. Keith Conners of the University of Pittsburgh, that were made known at the hearing:

The Food and Drug Administration has promised to make recommendations shortly on where to go from here. That is the least the agency can do. It is regrettable that the FDA has not taken a position of leadership in this crucial health issue, rather than lagging behind until all but forced to action because of public opinion. As for the manufacturers of artificial foods, little can be expected of them except business as usual. The burden of proof in these matter seems to rest upon those who believe a substance is dangerous rather than those, such as the manufacturer, who claim that it is safe. The effect of this attitude, in the case of fake flavors and colors consumed by children, is to make guinea pigs of our children and laboratories of our homes.⁷²³

By basing his/her comments on Conners' trial, which had not yet been peer-reviewed, let alone published, by the time the story was published, the author demonstrated how both Feingold's supporters and his detractors were guilty of exaggerating or extending the findings of the clinical trials of the Feingold diet in ways that supported their own views. Despite the fact that Senator Edward Kennedy stated during the Hearings that Conners' result 'probably isn't conclusive', the editorial insisted that the psychologist's findings indicated that 'the need is now for immediate and expanded testing that will show either that a problem exists or it does not'.⁷²⁴ In this way, the approach taken by the author of this positive editorial was not so different from that of the Nutrition Foundation which reported negatively on the diet prior

⁷²³ Anonymous, 'Children and Artificial Food', *Washington Post*, 14 September 1975, 34.

⁷²⁴ Edward Kennedy quoted in Anonymous, 'Children and Artificial Food', 34.

to the publication of any controlled trials, and believed that there was little need for such ‘expanded testing’.⁷²⁵

Other writers were quick to place a fair amount of credibility in Feingold’s claims, even though clinical trials into his hypothesis had not been completed. Nicholas von Hoffman, (b. 1929), for example, used Feingold’s hypothesis to criticise Attorney General William Saxbe’s argument that increase in crime was due to ‘parents, permissiveness and pornography’.⁷²⁶ Stating that ‘neither the conservative attribution of crime to pornography nor the liberal’s blaming it on bad housing show a convincing chain of causality’, von Hoffman wondered if the increase in crime ‘may be traceable to the involuntary ingestion of drugs in our food supply’.⁷²⁷ Von Hoffman proceeded to posit that Feingold’s ‘hypothesis would also explain the correlation between crime and family income. It’s lower income people who can’t afford fresh, unadulterated food and whose social surroundings don’t frown on the consumption of cellophane-wrapped Blinky-Tinkies and all the other chemically manufactured junk foods’.⁷²⁸ Although von Hoffman’s political leanings were more ambiguous than those of Colman McCarthy, his article, written months before *Why Your Child is Hyperactive* was published in late 1974, indicated how Feingold’s hypothesis could be used to critique many aspects of American society and government.

For Robert Rodale (1930-1990), the son of health food publishing magnate J. I. Rodale and, after the senior Rodale’s death in 1971, the head of Rodale Inc., Feingold’s theory had both political and financial relevance.⁷²⁹ Rodale’s syndicated column, ‘Organic Living’, appeared

⁷²⁵ See Chapter 5.

⁷²⁶ Nicholas von Hoffman, ‘Concerning Hyperkinesis, Food Additives and Crime’ *Washington Post*, September 11, 1974, B1, B6, at p. B1

⁷²⁷ *Ibid.*, B1, B6.

⁷²⁸ *Ibid.*, B6.

⁷²⁹ Robert Rodale’s death was not as famous as his father’s who died during a taping of the *Dick Cavett Show* (see above), but it was still cruelly ironic. One of J. I. Rodale’s boasts was ‘I’m going to live to be 100 unless I’m run down by a sugar-crazed taxi driver’, refined sugar being one of the foods Rodale condemned; his son was sadly killed at the age of sixty in an automobile accident in Moscow. Greene, ‘Guru of the Organic Food

in numerous American newspapers and, given that his publishing business depended partly on dissatisfaction with the food supply, Rodale had a clear financial incentive to support Feingold's claims and did so using strong language. Rodale described the food industry's use of additives as 'possibly the most heinous "crime" perpetrated by the food processors on an unknowing public', a crime that affected children the most.⁷³⁰ Continuing on this theme, Rodale speculated:

Let's imagine, and it doesn't take too much, that Dr. Feingold's theories are proven correct. Does this mean that food processors can be tried for the 'crime of negligence' or can food processors who cause cancer be tried also? Maybe they should, if only in the court of public opinion.⁷³¹

Another article by Rodale which mentioned the Feingold diet emphasised how scientists were unable to predict or explain the potentially hazardous effects of the '2,500 substances currently being added to our food supply', and that the only way to be safe was to avoid additives altogether.⁷³² Rodale began by discussing how 'a strange thing happened recently in a University of West Virginia laboratory when adult house flies were fed a diet containing common food coloring additives. As soon as the flies were exposed to light, they died.' A picture of a dead fly and a vial of dye illuminated by a bright light accompanied the story. According to the postdoctoral research fellow who observed the phenomenon, 'the flies were killed by photodynamic action, a destructive effect produced when the dye and normal light interact'. The research fellow added ominously that 'the wide usage of dye additives in foods, drugs and cosmetics, could result in photodynamic injury to man'.⁷³³ Rodale proceeded immediately to describe Feingold's findings about food additives and

Cult', 56; Glenn Fowler, 'Robert Rodale, 60, Dies in Crash; Publisher Backed Organic Farms', *New York Times*, 21 September 1990, www.query.nytimes.com/gst/fullpage.html?res=9C0CE7D71531F932A1575AC0A966958260, accessed 4 August 2008.

⁷³⁰ Robert Rodale, 'Can Pure Food Be a Reality?', *Hartford Courant*, 26 February 1975, 49.

⁷³¹ Ibid.

⁷³² Robert Rodale, 'Food Additives Sometimes Cause Strange Effects', *Hartford Courant*, 29 May 1974, 23.

⁷³³ Ibid.

hyperactivity, as well as similar observations made by Pennsylvania physician Stephen D.

Lockey:

Many instances of mild psychological trauma and other weird symptoms could conceivably be caused by food chemicals. But scientists may never be able to pinpoint which ones are responsible, because of the complex interactions between all the thousands of chemicals in our environment. While researchers are busy trying to solve the puzzle, you can protect yourself by eating foods that don't contain additives.⁷³⁴

Although Rodale's business interests meant that readers might greet his concern about food additives with scepticism, the observations of investigative reporter Morton Mintz were more difficult to dismiss. Not only was Mintz a respected journalist for the *Washington Post*, he had also been first to report on a number of health-related scandals, most notably the thalidomide disaster in 1962.⁷³⁵ Unlike those by McCarthy and Rodale, the tone of Mintz's front page story was measured, and he refrained from making judgements about Feingold's theory. Mintz added how hyperactivity could also be treated with amphetamines such as Ciba-Geigy's Ritalin, mentioning that the drug 'accounted for \$11 million in sales', but did not suggest explicitly that the Feingold diet might jeopardise such profits.⁷³⁶

Despite the somewhat muted tenor of Mintz's article, it quickly generated significant attention. According to Colman McCarthy, who worked with Mintz at the *Washington Post*, Mintz's story on the Feingold diet resulted in more mail being sent to the reporter than on any other subject he had covered in twelve years, when he had first written about the thalidomide disaster.⁷³⁷ Senator Glen Beall, Jr. (1927-2006) of Maryland was so impressed by the article that, a day after it was published, he added a copy of it, as well as a speech Feingold gave in London, to the Congressional Record.⁷³⁸ Such a response was likely due in part to Mintz's

⁷³⁴ Ibid.

⁷³⁵ Morton Mintz, "'Heroine' of FDA Keeps Bad Drug Off of Market', *Washington Post*, 15 July 1962, A1-A2.

⁷³⁶ , A1, A9.

⁷³⁷ McCarthy, 'Color it Dangerous', C9.

⁷³⁸ United States Congress, *USA Congressional Record*, S1936-19742.

previous successes in breaking the thalidomide story, but it also reflected that the fact that public was concerned about the hidden effects of food additives on health.

While Mintz's penchant for investigative health journalism spurred him to write about the Feingold diet, other writers gravitated towards the Feingold diet because of their interest in the health food industry or holistic medicine. Writer Tom Monte, for example, described how he quit his job as a newspaper reporter in the mid-1970s because his paper refused to publish a story about macrobiotic diets.⁷³⁹ He proceeded to edit *Nutrition Action*, the journal of the Center for Science in the Public Interest (CSPI), and then became a freelance writer and lecturer specialising in complementary health and macrobiotic nutrition.⁷⁴⁰ Monte's story, 'Feingold Diet: A Precious Commodity to Hyperactive Children?', was a clear endorsement of Feingold's hypothesis.⁷⁴¹ He began by describing a Halloween party for hyperactive children in Maryland, and expressed his surprise at how calmly the thirty children present behaved. The explanation for their good behaviour was the Feingold diet. According to Monte, 'every Feingold member I spoke to at this Halloween party and in later interviews reported remarkable stories about the improved behavior of their children once they began the Feingold diet'.⁷⁴²

As with many articles that provided support for the Feingold diet, testimonials from families were a compelling aspect of Monte's story. Such accounts not only provided anecdotal evidence to support Feingold's theory, but they also gave hope to families who were desperate to improve the behaviour of their children. One family that Monte described, the Johnsons, had been told by their physician 'that their son Brian, then 7, would be

⁷³⁹ Macrobiotic diets, or 'long life' diets stress local, whole and unprocessed foods, with grains consisting of at least fifty per cent of caloric intake.

⁷⁴⁰ The information about Tom Monte can be found on www.tommonte.com/about.html (accessed 14 August 2008).

⁷⁴¹ Monte, 'A Precious Commodity?'

⁷⁴² *Ibid.*, W11.

institutionalized because of his disruptive behavior'. After a semester on the Feingold diet, however, 'Brian's grades went from C's to A's. He was taken off the Ritalin and was no longer a disruptive force in school, that is, so long as he avoided artificial colors and flavors'.⁷⁴³

A story in the *Los Angeles Times* also described how the Feingold diet could result in remarkable transformations. As journalist Marlene Cimons explained:

It took a long time for Mina Otis to find out why her child was uncontrollable. Each new doctor had a different theory. 'We were told he was allergic,' she said. 'We were told he was a screwball.' Raymond Ellis Otis, 11, was neither. He was one of an estimated 5 million children in this country who are hyperactive ... Until this past July, Mrs. Otis had no idea what to do about her son's erratic behavior. 'But now when he wakes up in the morning, his hair isn't all scruffed up from tossing and turning,' Mrs. Otis said. 'He doesn't grind his teeth. When you ask him a question, you get a paragraph answer instead of an "I don't wanna."' He is able to concentrate.' She looked at her son. 'And he doesn't do anything dumb,' she said. ... The change in Raymond Otis, his mother said, finally occurred after she put him on a diet free of artificial colors and flavors and free of foods containing natural salicylates.⁷⁴⁴

Mina Otis proceeded to apologise to Cimons for the current behaviour of her son; he had mistakenly eaten some corn with artificially coloured butter on it. In addition, Cimons criticised the use of amphetamines to treat hyperactivity, relating the story of a lawsuit filed on behalf of seventeen children from Taft, California which alleged 'that school officials forced them to take Ritalin, which, in at least one case, resulted in an epileptic seizure'.⁷⁴⁵ By contrasting the Otis' story with that of the lawsuit, Cimons insinuated that the Feingold diet was a tool that allowed parents to wrest control over their child's health back from authorities such as the school board and the medical profession. In this way, it not only served its purpose as a therapy, it also empowered parents such as Mina Otis.

⁷⁴³ Ibid., W12

⁷⁴⁴ Marlene Cimons, 'Hyperactivity and Food Additives', D1.

⁷⁴⁵ Ibid., D6.

The theme of empowerment was also featured in *Utah Holiday*, a small magazine which described two families which had successfully employed the Feingold diet. Dee and Lavon Seely had adopted three hyperactive children from another state. Their physician had recommended corporal punishment but this was not effective and ‘after a while the neighbors began to wonder what was happening in the Seely home. . . . Finally, one neighbor reported them for child abuse’.⁷⁴⁶ The Seelys also tried Ritalin, but ‘the Ritalin would wear off in two hours and then the children would be even wilder’. Eventually Lavon found out about the Feingold diet and, within three days, her most disruptive child’s behaviour was improving.⁷⁴⁷ Cleo Jeppson, the mother in the other family described by Jarvik, explained how, once her daughter Lisa started the Feingold diet, ‘it was like someone peeled off an outer layer and for the first time I saw my daughter as herself . . . I wanted to stand on the roof and shout to the world’.⁷⁴⁸ Jeppson was so impressed that she founded the Feingold Association of Utah.

‘Another “Miracle” Diet Cure that Failed’?

Although most newspaper stories during the 1970s were favourable towards the Feingold diet, there were articles that reported on the findings of clinical trials that were negative and editorials by columnists who were sceptical of Feingold’s hypothesis. Editorials that rejected the Feingold diet were typically written by physicians and scientists who had previously expressed little sympathy for concerns about food additives generally. One such critic was Harvard nutrition scientist Frederick Stare, who attacked Feingold’s hypothesis from many angles in his ‘Food and Your Health’ column. For example, Stare argued that Feingold had ‘not reported his results in any recognized scientific journal so that other professionals can evaluate his methods and results’, and charged that this was ‘not only irresponsible but a

⁷⁴⁶ Elaine Jarvik, ‘The Calming’, 50.

⁷⁴⁷ Ibid.

⁷⁴⁸ Ibid., 49.

source of concern because a proscription against additives and particular types of food would lead to the avoidance of a number of common sources of important nutrients'.⁷⁴⁹

Although Feingold was able to refute both of these arguments, and did so in a speech to the Newspaper Food Editors and Writers Association in 1977, he was less able to refute another one of Stare's charges.⁷⁵⁰ This was that the Feingold diet only elicited a placebo effect. Stare explained that:

Unfortunately, the special diet is so drastic (no soft drinks, candy, bakery goods, ice cream, jellies and jams and so forth) that many aspects of family life undoubtedly change as a result. Accordingly it is possible that children's behavior may change (or parents' appreciation of children's behavior may change) as a result of increased family 'togetherness' while the result is attributed to the elimination diet. There is no way at present to be certain that the good results apparent in Dr. Feingold's anecdotes are in fact more than a placebo effect due to his own enthusiasm and conviction.⁷⁵¹

Stare's suggestion that a child's improvement on the Feingold diet was merely an example of placebo effect became one of the most common arguments levelled against Feingold, and one that was often repeated in media reports. The media typically reported arguments about placebo without questioning what this precisely meant, or what the placebo effect actually represented. One reason for this may be that, as historian Anne Harrington has described, placebo was the topic of much scientific discussion during the 1970s and 1980s, with researchers such as psychiatrists Arthur Shapiro (1922-1995), Jerome Frank (1910-2005) and Robert Ader exploring how the doctor-patient relationship and other situational factors could affect healing.⁷⁵² Although Feingold would retort in response to these claims that there 'may be an element of placebo, but the whole practice of medicine is placebo', contemporary interest in placebo meant that there was weight to such arguments within the media and in

⁷⁴⁹ Frederick Stare, 'Placebo Effect No Substitute for Research', *Hartford Courant*, 27 March 1975, 36;

Frederick Stare, 'Do Additives Make Your Child Hyperactive?', *Hartford Courant*, 30 December 1975, 8.

⁷⁵⁰ Feingold, 'A View from the Other Side', 8-12.

⁷⁵¹ Stare, 'Placebo Effect', 36.

⁷⁵² Anne Harrington, 'Introduction' in Anne Harrington (ed.), *The Placebo Effect: An Interdisciplinary Exploration* (London: Harvard University Press, 1997), 1-11, at pp. 2-8.

medical literature.⁷⁵³ In a way, such critiques were reminiscent of those made of food allergists during the post-war period which stated that the symptoms of food allergy were chiefly psychosomatic. Feingold had written about psychosomatic allergy himself, but believed that psychosomatic factors were ‘contributory rather than primary’, in effect, exacerbating the responses to allergens, instead of replacing them as the primary cause of allergic symptoms.⁷⁵⁴

The discovery of endorphins, hormones known for their analgesic and euphoria-inducing effects, during the mid-1970s also contributed to interest in placebo, since they were found to play a role in certain types of placebo effect. As Harrington states, linking endorphins with placebo meant that ‘placebo, an “imaginary” treatment, had been found to have some solid flesh on its bones after all’.⁷⁵⁵ Newspapers picked up on scientific interest in placebo, and reported regularly on both its potential importance to medicine and how it could explain the effectiveness of contentious treatments such as acupuncture.⁷⁵⁶

Increased scientific respect for the placebo effect did not, however, help Feingold or his followers persuade physicians and food manufacturers that food additives were harmful. In fact it did the opposite. According to a Nutrition Foundation committee that reviewed studies of the Feingold diet in 1980:

successes reported by parents of children given an additive-free diet were most likely caused by a “placebo effect” where the power of suggestion and hope actually produces the desired response. . . . Since the food additive-free diet has no apparent harmful effects, and since the non-specific (placebo) effects of this dietary treatment are frequently very beneficial to families, we see no reason to

⁷⁵³ Feingold quoted in Monte, ‘A Precious Commodity?’, W12.

⁷⁵⁴ Feingold, ‘Treatment of Allergic Disease’, *JAMA*, 323; Feingold, *et al*, ‘Psychological Studies’, 195, 198, 201.

⁷⁵⁵ Harrington, ‘Introduction’, 4-5.

⁷⁵⁶ Lawrence K. Altman, ‘Physicians Urged to Widen Understanding of Placebo’, *New York Times*, 2 July 1975, 19; Boyce Rensberger, ‘Acupuncture Likened to Placebo’, *New York Times*, 19 June 1975, 37.

discourage those families who wish to pursue this type of treatment as long as they continue to follow other therapy that is helpful.⁷⁵⁷

While the Nutrition Foundation might have accepted reluctantly that the Feingold diet was harmless, and that it might even inadvertently help families by virtue of its placebo effect, such conclusions nevertheless reinforced their claim that food additives had nothing to do with hyperactivity. Indeed, Morris Lipton, the chair of the Nutrition Foundation's investigation of the Feingold diet, charged that, far from being hazardous, food additives were a fundamental element of Western society. In an article in which Lipton's assessment of the Feingold diet was juxtaposed against those of Senator George McGovern, psychologist C. Keith Conners and Feingold himself, the psychiatrist asked: 'Where would our society be without food preservatives? The shelf life of bread would be eight hours; there would be no ham, bacon, sausages, fresh vegetables, etc. How would we feed the millions in our cities? Lest we forget: Columbus discovered America seeking food preservatives and spices.'⁷⁵⁸

Lipton's comments were somewhat misleading, since he only mentioned artificial preservatives, and not the synthetic colours and flavours that Feingold also targeted.

Companies did not use these additives to safeguard the food supply, but rather to improve the marketability of their products. As Earl M. Handing, a marketing manager for food chemical company Warner-Jenkinson, stated in a 1976 *Los Angeles Times* story about food dyes:

'Cosmetic effect is most important and it gives the competitive edge to those foods with the most appealing color ... People don't want gray-colored hot dogs and sausages ... Also, how would you distinguish different flavors in gelatins all the same color?'⁷⁵⁹ The need for bread that lasted longer than eight hours being more pressing than the need to distinguish

⁷⁵⁷ Nutrition Foundation quoted in Al Rossiter, Jr., 'Feingold Diet Disputed', United Press International, 16 October 1980.

⁷⁵⁸ Morris Lipton, 'Can Food Chemical Additives Have Any Effect on Behavior?', *The Hartford Courant*, 3 August 1977, 22.

⁷⁵⁹ Anonymous, 'The Importance of Food Coloring', *Los Angeles Times*, 24 June 1976, 30.

between artificially flavoured Jell-O products, Lipton rather sensibly limited his line of argumentation to artificial preservatives.

Nonetheless, Lipton's arguments about the necessity of artificial preservatives were similar to those levelled against Rachel Carson a decade earlier with regards to pesticides, namely, that American society had grown dependent upon pesticides and would face a food supply disaster if the government restricted them. By delineating such harrowing scenarios, defenders of food additives were, in part, matching the dire speculations made by Carson, Feingold and others with respect to what would occur if such substances remained in the food supply. As Feingold asserted in his section of the article: 'Poor nutrition is now being closely scrutinized as a cause of juvenile delinquency, vandalism in schools and learning disabilities.'⁷⁶⁰ While Feingold's statement may not have been as polemical as Lipton's - Feingold stated that poor nutrition was 'a' cause, rather than 'the' cause, and referred to poor nutrition generally, rather than food additives specifically - he had written at length in *Why Your Child is Hyperactive* about how chemicals were causing an increase in anti-social behaviour, as well as hyperactivity.⁷⁶¹ As with the debates about food additives during the late 1960s and early 1970s, fear-mongering based on often unsubstantiated speculation was a rhetorical strategy employed by both sides of the Feingold debate.

Criticism of the Feingold diet, however, did not have to be as heavy-handed as the pieces by Lipton or Stare to be effective. When asked in a letter about whether there was 'truth to claims that food additives cause hyperactivity' in his *Chicago Tribune* health advice column, physician G. Timothy Johnson (b. 1936) answered without invective, but also managed to mention the difficulty of maintaining the diet, Feingold's lack of scientific evidence, the

⁷⁶⁰ Ben F. Feingold, 'Can Food Chemical Additives Have Any Effect on Behavior?' *The Hartford Courant*, 3 August 1977, 22.

⁷⁶¹ Ben F. Feingold, *Why Your Child Is Hyperactive* (New York: Random House, [1974] 1996), 156-68.

paucity of supportive trials and a review of research into the diet found in the Nutrition Foundations's mouthpiece *Nutrition Reviews*. Responding to the writer's concerns about Ritalin, Johnson stated, 'I can understand your reluctance to have your child take a drug, but I would remind you that stimulant drug therapy is a time-tested treatment.'⁷⁶² In the process, Johnson not only listed many of the criticisms of the Feingold diet, but also insinuated that the effectiveness of stimulant drugs precluded even the need for an alternative approach to hyperactivity.

A little over a year later, Johnson addressed the Feingold diet again, stating that it was 'obvious the fuss over food additives being a possible cause of hyperactivity in children will not disappear quickly. I receive many letters on the subject, and pediatricians tell me parents often ask about it.'⁷⁶³ Despite continued interest in the Feingold diet, Johnson downplayed the link between food additives and hyperactivity, assuring his readers that most 'experts believe that if a relationship exists between diet and behavior, it is of relatively minor importance or exists only within a small subpopulation of children'. Although he believed 'the question deserves further study', the somewhat exasperated tone of his article, not to mention its title, 'Diet-Hyperactivity Link Still Unproved', made his opinion about the Feingold diet clear.⁷⁶⁴

'On Nutrition' and the Feingold Diet

Other health columnists were not as decided, however, about whether the Feingold diet was a viable alternative for hyperactive children or not. The best example of this was the 'On Nutrition' column which began in 1976 and was written by nutrition scientist Jean Mayer and his colleagues Joanna Dwyer and Jeanne Goldberg, with whom he worked at Harvard and

⁷⁶² Johnson was and continues to be ABC Television's medical editor. Johnson, 'Food Additive Link', *Chicago Tribune*, A13.

⁷⁶³ Johnson, 'Diet-Hyperactivity Link', A5.

⁷⁶⁴ Ibid.

then Tufts Universities. One might have suspected that the Franco-American Mayer, who had significant connections to the food industry and had been a colleague of Frederick Stare, would have been highly sceptical of Feingold's hypothesis.⁷⁶⁵ Indeed, an early newspaper feature pitted Mayer against Feingold in a debate about the danger of food additives.⁷⁶⁶ But even this column reveals that Mayer understood that the issue of food additives and health was complicated.

Although Mayer concluded his half of the debate by stating that people were 'more likely to be run over by a car than you are to be killed or harmed by an additive', the bulk of his article related to the difficulty in assessing the risk of food chemicals on human health.⁷⁶⁷ This was primarily because, as Mayer described, 'we are morally opposed to testing possibly poisonous substances on human beings'. To get around this moral hurdle, scientists used animals to test food chemicals, but 'no matter how careful the food industry and the agencies are, some intellectual and practical problems remain', namely, that some substances, such as Vitamin D, needed to be taken in near toxic doses to be effective and that 'the metabolic rate varies from species to species and compound to compound'. In other words, it was difficult

⁷⁶⁵ Although Mayer had worked with Stare, their relationship had hardly been amiable and contributed to Mayer's departure to the less prestigious Tufts University. According to an anonymous interviewee: 'It was said that Jean Mayer was so opposed to what Stare was doing so that if he met him in a hallway, he would duck into a room so as not to encounter Stare. He just felt that he was a prostitute. And that was why Mayer went to Tufts, because he realised he would never be able to get up on the ladder at Harvard as long as Stare had the influence there.' Historian Harvey Levenstein has substantiated this, stating that Mayer and Stare did not agree on many issues. Given Mayer's impressive wartime record with the French Free Army and the French Resistance, as well as his ability to 'stand up to and face down the most persistent of critics', the differences with Stare must have been strong indeed. Harvey Levenstein, *Paradox of Plenty: A Social History of Eating in Modern America* (Oxford: Oxford University Press, 1993), 155; Anonymous, In-person interview; 9 December 2007.

⁷⁶⁶ Ben F. Feingold, 'The Arguments Con', *Washington Post*, 19 September 1974, F1, F10; Jean Mayer, 'The Arguments Pro', *Washington Post*, 19 September 1974, F1, F10.

⁷⁶⁷ Mayer's comment about being more likely to be run over by a car were somewhat odd, as Feingold himself observed, given that in 1974, the year in which his article was published, over 45 000 Americans were killed in motor vehicle accidents. Incidentally, the figure in 1973 was over 54 000 fatalities, marking the second deadliest year on record, 1972 being the worst ever year. As Feingold noted, food chemical industry spokesman Arthur T. Schramm had similarly stated in 1956, the 'most impressive example of man's willingness to expose himself to hazards for a convenience is his continued use of the automobile. Were the automobile considered in the same manner as certified colors, the shocking toll of 47, 000 killed in 1956 would certainly demand delisting'. Advocates for Highway and Auto Safety, www.saferoads.org/federal/2004/TrafficFatalities1899-2003.pdf accessed 24 September 2008; Feingold, 'The Arguments Con', F10; Mayer, 'The Arguments Pro', F10.

to apply results from animal trials to humans and also to extend findings about one particular substance to another:

at high levels, particularly when injected, MSG [monosodium glutamate] causes considerable damage to animals. It can destroy certain eye and brain cells and result in abnormal growth patterns. We use MSG at much lower levels and we don't inject it into ourselves. However, since mice metabolize excess MSG at 30 times the rate for humans, it takes comparatively larger doses to build up a toxic level in mice. Given this difference and the fact that some people become quite sick when they absorb MSG (especially in clear soup on an empty stomach) can we still use the standard formula of one-hundredth the amount that causes damage in animals, to determine 'safe' doses of MSG?⁷⁶⁸

Mayer proceeded to advise readers not to 'worry unduly about additives', but also that:

Without more perfect methods of testing the best advice I can give is, use your common sense. I would use any foods about which I had doubts in strict moderation and, whenever possible, use fresh foods. They are better both nutritiously and in terms of taste. Politically, I would let your U.S. senator and representative know that you want the government's regulatory bodies to be able to investigate and enforce safety measures.⁷⁶⁹

Mayer's words amounted not so much to a defence of food additives as a word of caution about how much faith consumers should have in nutrition science, no matter whether it reflected well or poorly on food additives. Feingold, while emphasising the validity of his observations of the effects of food additives on children, did not necessarily disagree with Mayer's assessment of evidence from animal testing, but instead suggested that 'the use of any compound whether as a drug or as a food additive must be determined on the basis of benefit compared with risk'. Although his view would change by 1977, Feingold explained in his response to Mayer that food preservatives, for example:

are essential to our food supply. Without preservatives our entire system of food distribution would collapse. Fortunately, adverse reactions to most preservatives seem to occur rather infrequently, which justifies their continued use. However, even these compounds should be under constant surveillance while research continues for better compounds. The experience with food colors is just the opposite. ... the colors are not essential: they have no nutritional value. Their sole function is a cosmetic. Without them, nothing

⁷⁶⁸ Mayer, 'The Arguments Pro', F10.

⁷⁶⁹ Ibid.

would be lost. In other words, in evaluating the synthetic colors, risk far outweighs the benefits.⁷⁷⁰

Established as a debate about the pros and cons of food additives, the opinions expressed by Feingold and, especially, Mayer underscored instead the complexities of the food additives dilemma. The chief difference between their positions was not so much that food additives could be dangerous, but what to do about additives that did pose a threat. While Feingold's experience as a clinician convinced him that fairly drastic action was warranted, Mayer's hesitance was perhaps due to his intimate knowledge of and connections to how the food industry operated, and the reliance his then employer, Harvard's Department of Nutrition, had on corporate funding. Nevertheless, Mayer's subsequent columns about the Feingold diet, first co-authored by Joanna Dwyer and, later, by Jeanne Goldberg, demonstrated that he continued to wrestle with the issue amongst the cacophony of conflicting reports and polemical arguments.⁷⁷¹

The first of these columns, written in August 1976 when the first controlled trials of the Feingold diet were still underway, identified many challenges inherent in determining whether or not Feingold's theory was a viable alternative for treatment of hyperactivity. The first problem noted by Mayer and Dwyer involved defining the terms involved in the debate. Not only were there more than 3000 food additives being used in the food supply, but the necessity of some was also deemed to be more significant than others. As Feingold had intimated in his 1974 debate with Mayer, food preservatives served a more vital role in the food supply than did artificial colours and flavours, which were employed primarily to

⁷⁷⁰ Feingold's lack of concern about preservatives prior to 1977, as well as other processed foods, such as refined sugar, was one of the reasons that he failed to gain much tangible support from the clinical ecology movement. Many supporters of clinical ecology believed that he did not go far enough in identifying dangerous additives. Feingold, 'The Arguments Con', F10; Anonymous, In-person interview, 9 December 2007.

⁷⁷¹ These columns were published in approximately 100 American newspapers. Anonymous, Email Interview, 10 December 2007.

enhance the marketability of processed foods and/or reduce the cost of their manufacture.⁷⁷²

Mayer and Dwyer echoed Feingold's notion, stating that while preservatives were 'necessary', food colours and flavours were 'only added to a food to make it look or taste better'.⁷⁷³ One had to distinguish between different types of food additives, therefore, in order to analyse whether their benefits outweighed their risks.

Mayer and Dwyer also warned that the definition of the hyperactive child was difficult to determine. There was 'no precise definition of hyperactivity. What may be a hyperactive child in the eyes of some parents or teachers is a normal, high-spirited child in the eyes of others.'⁷⁷⁴ This was despite the fact that the authors also claimed that 'true hyperkinesis' was proven if a hyperactive child responded positively to stimulants such as Ritalin, a notion which psychiatrists had begun to question.⁷⁷⁵ Although the authors did not pursue how such 'complicating factors' might affect the research intended to determine the validity of the Feingold diet, the fact that the two primary terms of reference concerning the diet were imprecise suggested that debates about Feingold's hypothesis would be difficult to resolve.⁷⁷⁶

Subsequent columns by Mayer and Dwyer reinforced the complications inherent in drawing conclusions about the diet. In a November 1977 column, for example, the authors responded to a question from a reader about whether or not trial results were providing support for Feingold's hypothesis. Mayer and Dwyer responded that evidence from the trials had been inconclusive thus far because 'support for the effectiveness of this elimination diet comes mainly from what children and their parents themselves say' and that 'studies designed to

⁷⁷² Feingold, 'The Arguments Con', F10.

⁷⁷³ Jean Mayer and Joanna Dwyer, 'Food Additives, Hyperactivity and Dr. Feingold's Diet', *Washington Post*, 5 August 1976, F3.

⁷⁷⁴ Mayer and Dwyer, 'Dr. Feingold's Diet', F3.

⁷⁷⁵ Paul L. Adams, 'Review of, *Hyperactivity: Research, Theory, and Action* by Dorothea M. Ross and Sheila A. Ross, *American Journal of Psychiatry* 134 (1977), 833-4.

⁷⁷⁶ Mayer and Dwyer, 'Dr. Feingold's Diet', F3.

test the theory were not well-controlled enough to permit any objective conclusion'.⁷⁷⁷ As Chapter 9 demonstrates, these two issues would often plague researchers trying to draw conclusions about the Feingold diet.

With respect to anecdotal reports of the Feingold diet, patient and parental descriptions of their experiences with the diet would continue to be viewed with scepticism by most medical researchers and scientific observers. Mayer and Dwyer's dismissal of what children and parents had to say about the Feingold diet was reinforced at the end of their column when they reiterated the AAP's assertion that parents should not attempt the diet because of fears about its 'long-term effects', specifically, regarding the diet's elimination of certain fruits during the early stage of the diet, and the idea that some children might interpret the diet as punishment.⁷⁷⁸ Such beliefs highlighted the assumption that parental observations and patient experiences were irrelevant in assessing the effectiveness of the Feingold diet, in particular, and in the evaluation of child health generally, and that clinical trials were the only meaningful arbiters of novel medical ideas. The emphasis on the power of clinical trials to resolve medical controversies, however, placed a great deal of faith in a process that was far from perfect. As Mayer and Dwyer admitted, not only were the trials designed to test the Feingold diet complicated to control, but it was also difficult to determine which food additives were to be tested and how to assess the improvement of a child's behaviour. Although they were confident that subsequent trials would be better controlled, they also cautioned that it would 'be some time before a scientifically-valid assessment of the theory ... is available'.⁷⁷⁹ Perhaps aware of their somewhat mixed message, the nutritionists concluded by suggesting that 'cutting down on additives and eating foods that are fresh or very lightly processed is a good idea'.⁷⁸⁰

⁷⁷⁷ Jean Mayer and Joanna Dwyer, 'Nutrition', *Washington Post*, 17 November 1977, F28.

⁷⁷⁸ Mayer and Dwyer, 'Dr. Feingold's Diet', F3; Mayer and Dwyer, 'Nutrition', F28.

⁷⁷⁹ Mayer and Dwyer, 'Nutrition', F28.

⁷⁸⁰ *Ibid.*

The next two columns by Mayer and Dwyer, written a week apart in November 1978, made matters even more complicated. The first column, titled ‘Diet Changes Seem to Help’, seemingly suggested that evidence had emerged which supported Feingold’s theory, but the column’s contents, as well as the one which followed it, revealed a murkier picture.⁷⁸¹ The nutritionists reported on the research conducted by a group at the University of Toronto which compared the effect of the Feingold diet with that of stimulant medication in reducing the hyperactivity of twenty-six children.⁷⁸² Although the researchers determined that stimulant medication was more effective, they agreed with Feingold that dietary changes appeared to work, especially when in combination with stimulant drugs. In attempting to explain why this was the case, however, Mayer and Dwyer added another factor to the equation, namely, the possibility that it was sugar, not additives, that was the causative factor in some cases of hyperactivity.⁷⁸³

Sugar had been suggested as a possible cause of hyperactivity in both the popular media and medical literature.⁷⁸⁴ Never attaining the popularity of the Feingold diet, blaming sugar for hyperactivity nevertheless appealed to many parents, and nutritionists such as Mayer and Dwyer, even when research suggested that there was no such link.⁷⁸⁵ With regards to the

⁷⁸¹ Jean Mayer and Joanna Dwyer, ‘Diet Changes Seem to Help’, *Chicago Tribune*, 16 November, 1978, D34. Other stories about the Feingold diet had headlines that were misleading as to the actual content of the story. One Associated Press article, entitled ‘Report Sees No Relationship Between Hyperactivity, Diet’ nevertheless expressed the views of both FAUS and the Nutrition Foundation and concluded with the words of Sanford Miller, director of the Foods division of the FDA, that ‘the jury is still out on the question’. Warren E. Leary, ‘Report Sees No Relationship between Hyperactivity, Diet’, *Associated Press*, 16 October 1980.

⁷⁸² Jean Mayer and Joanna Dwyer, ‘Diet Changes’, D34.

⁷⁸³ Although Mayer and Dwyer’s suggestion about sugar was based on research done not by the University of Toronto group, but another research group, which they did not identify, but was likely one led by Richard J. Walsh of the New York Institute for Child Development, the fact that they seemed to approve of the findings of both groups is odd. This is because the Toronto group tested their subjects with chocolate cookies. While the test group received cookies containing food dyes, the other group ate dye-free cookies. Both the test and the control cookie, however, would likely have contained sugar, presumably throwing into doubt, at least according to Mayer and Dwyer, any evidence about the role of colours. Mayer and Dwyer, ‘Diet Changes’, D34; Carey Winfrey, ‘A Controversial Theory Links Hyperactivity to Nutrition’, *New York Times*, 14 January 1980, A15.

⁷⁸⁴ Winfrey, ‘Controversial Theory’, A15

⁷⁸⁵ Jane E. Brody, ‘How Diet Can Effect Mood and Behavior’, *New York Times*, 17 November 1982, C1.

Feingold diet, however, the proposition that there was a link between sugar and hyperactivity not only complicated what was already a confused debate, but also demonstrated how Feingold's desire to devise a practical treatment for hyperactivity altered the diet itself.

According to an anonymous correspondent of his, Feingold was cognizant that his diet had to appear as palatable as possible in order for it to work. Although he/she was able to convince Feingold during the mid 1970s that the common preservatives BHA (butylated hydroxyanisole) and BHT (butylated hydroxytoluene) could cause hyperactivity, and should be added to the elimination diet's list of banned substances, he/she was not able to convince Feingold that refined white sugar should also be added to the list. This was not so much because Feingold doubted that it was a factor; he suspected that it was, but was concerned that families would have too much difficulty eliminating sugar from their diet.⁷⁸⁶ He also did not want to attract the wrath of the sugar industry which was then involved in the debates over cyclamates.⁷⁸⁷ As such, sugar continues to be allowed in the Feingold diet, in the belief that it is the synthetic additives in sugary foods that trigger hyperactivity, rather than the sugar itself.⁷⁸⁸ Nevertheless, many families on the Feingold diet have independently taken sugar out of their child's diet and believe that it is a contributing factor.⁷⁸⁹

If the suggestion that it was sugar, not food additives, that was the key factor in rising rates of hyperactivity was not enough to confuse Mayer and Dwyer's readers, the nutritionists'

⁷⁸⁶ According to this source: 'When I suggested that sugar should be added, he said, "Oh yes, I know, but I can only suggest a few things, otherwise people will not follow any of it." I think he was trying to be very pragmatic.' Anonymous, 'In Person Interview', 9 December, 2007.

⁷⁸⁷ In her review of food and nutrition during the 1970s, food writer Marion Burros referred to the activism of the sugar industry numerous times. She described that, in an attempt to win over journalists, 'the Sugar Association was worried about sugar's bad press and put on a program at the annual Newspaper Food Editors' Conference entitled "Exploding Myths Associated with Sugar"'. Marion Burros, 'Eating Well May Be the Best Revenge; The '70s: A Decade of Concern; Looking Back Through the Consumer '70s', *Washington Post*, 30 December 1979, B1.

⁷⁸⁸ Feingold Association of the United States, 'The Feingold Program', www.feingold.org/pg-faq.html, accessed 9 October 2008.

⁷⁸⁹ For example, Bonnie Kowaliuk, Telephone Interview, 5 November 2007; Sean Corr, Interview; Brian Rigg, Telephone Interview, 19 May 2008; Lynn Murphy, Email Interview, 24 July 2008.

following column was likely up to the task. Whereas the tenor and title of their previous column suggested that some trials were supportive of the Feingold diet, the tone of this column was significantly more negative. It is difficult to say what had occurred during that week to change the authors' opinions; while it is possible that it was only then that they discovered pertinent research, it is more likely, although impossible to substantiate, that they were chastised by Feingold's detractors for the optimistic tone of their previous column. Mayer and Dwyer briefly discussed the research conducted by J. Preston Harley's team in Wisconsin and that led by Conners in Pittsburgh and concluded that 'if diet and hyperactivity are linked, the relationship is either very slight or present only in a limited number of children'.⁷⁹⁰ Although they refused to say definitively that diet played no role whatsoever in triggering hyperactivity, and suggested that children should eat minimally-processed foods, they also emphasised that 'we are able to do more for these children than we could a few years ago, thanks to the judicious use of stimulant drugs' and that 'most hyperactive children outgrow the disorder'.⁷⁹¹

In one sense, these last two comments betrayed a lack of sensitivity and understanding about the families who had attempted the Feingold diet, often as a last resort because stimulants had not been effective and because they could not simply wait for their child to outgrow their intolerable behaviour. The comments also implied, however, that many medical observers were simply not interested in pursuing alternative treatments for hyperactivity. Unlike many of the families of hyperactive children, health professionals such as Mayer and Dwyer, as well as G. Timothy Johnson and others, were satisfied that prescribing stimulants was not only efficacious, but it was also an ethical practice. They did not seem to understand why parents were hesitant to see their children given a prescription for amphetamines. On the

⁷⁹⁰ Jean Mayer and Joanna Dwyer, 'The Latest Tally on Diets for Hyperactive Kids', *Chicago Tribune*, 24 November 1978, F9.

⁷⁹¹ Mayer and Dwyer, 'Latest Tally', F9.

other hand, the nutritionists' repeated suggestion that parents serve fresh, unprocessed foods implied that, while the efficacy, or even necessity, of the Feingold diet as a treatment for hyperactivity was in question, there were inherent, yet undefined, problems with a diet rich in food additives.

Mayer wrote three more columns about the Feingold diet, the last two co-authored by Tufts University colleague and dietician Jeanne Goldberg, instead of Dwyer. These columns, published in 1979, 1980 and 1984, continued to discuss the Feingold diet ambiguously, reflecting the mixed results that had emerged from the trials designed to test Feingold's hypothesis. In 1979 the authors were 'still not sure that diet is the answer' and, reflecting on trials conducted in Toronto, Michigan and New York in 1980, they stated that 'we wish we could say that the results were clear-cut, but they're not'.⁷⁹² Although the title of Mayer and Goldberg's 1984 column, 'Weighing the Feingold "Elimination" Diet on its 10th Anniversary' implied a more definitive assessment of the diet, the authors continued to be ambivalent. Accepting that the Feingold diet might help some children, Mayer and Goldberg cautioned that it was 'highly restrictive' and that many of the diet's success stories could 'probably be charged to the placebo effect'.⁷⁹³

Unlike the glowing endorsements of the Feingold diet written by McCarthy and Rodale and the stinging dismissals penned by Stare and Lipton, the indecisive columns by Mayer, Dwyer and Goldberg highlighted the difficulties inherent in making objective decisions about the validity of Feingold's hypothesis, and provided a more balanced interpretation of the research than most other accounts. Other columnists who wrote regularly about the Feingold diet,

⁷⁹² Jean Mayer and Joanna Dwyer, 'Diet May Help Hyperactive Children', *Chicago Tribune*, 9 August 1979, F24; Jean Mayer and Jeanne Goldberg, 'Hyperactive Children: When the Food Dye is Cast', *Los Angeles Times*, 28 August 1980, J42.

⁷⁹³ Jean Mayer and Jeanne Goldberg, 'Weighing the Feingold "Elimination" Diet on its 10th Anniversary', *Los Angeles Times*, 27 September 1984, SF40.

such as nutrition writer Jane E. Brody, also vacillated with regards to its efficacy. As Brody described in one of her later columns, one of the primary obstacles to getting clear answers was that there existed a ‘classic standoff between the plodding nature of rigorous scientific research and the public need for expedient answers to costly, distressing problems’.⁷⁹⁴

Journalists, such as Brody, who reported on the story of the Feingold diet throughout the decade when it was mainstream news, could not ignore that there were problems reconciling the anecdotal stories provided by parents and physicians and some of the findings reported by clinical investigators. Journalists or commentators who, instead, wrote overwhelmingly positive or negative accounts of the Feingold diet were simply not accounting for many of the factors involved in the debate.

Brody also warned, moreover, that there was ‘a price to pay for misapplication of scientific research’.⁷⁹⁵ She cited, for example, the case of the ‘Twinkie Defence’, which arose in the trial of Dan White (1946-1985) for the murder of the San Francisco mayor, George Moscone (1929-1978) and Supervisor (city councillor) Harvey Milk (1930-1978), the first openly gay man to be elected to public office in California.⁷⁹⁶ During the trial, Martin Blinder, a psychiatrist who testified for White, mentioned that the defendant’s consumption of Twinkies and Coca Cola (he had previously been a health food advocate), along with problems at home and at work (he had also been a San Francisco Supervisor, but had recently quit), contributed to his depression and, subsequently, diminished his responsibility for his actions. Although it is arguable how much a role the Twinkie defence actually played in the case, White was found to have diminished capacity and was only convicted of voluntary manslaughter, serving five years of his seven-year sentence before being released and committing suicide in

⁷⁹⁴ Jane E. Brody, ‘Diet Therapy for Behavior is Criticized as Premature’, *New York Times*, 4 December 1984, C1, C15, at p. C15.

⁷⁹⁵ Brody, ‘Diet Therapy’, C15.

⁷⁹⁶ *Ibid.*, C1. A documentary about Harvey Milk and his assassination, *The Times of Harvey Milk*, won the Oscar in 1985. A Gus Van Sant biographical film about Milk, called *Milk*, was also released in 2008 and actor Sean Penn won the Oscar for Best Actor for his portrayal of Milk.

1985.⁷⁹⁷ Moreover, journalists, such as Brody, picked up on the notion that certain foods could cause pathological behaviour and linked it directly to some of Feingold's hypotheses about food additives and anti-social behaviour. It was one thing to blame a child's hyperactivity on food additives; it was quite another to acquit murderers on the basis of the Twinkie defence.

⁷⁹⁷ Carol Pogash, "The Myth of the "Twinkie Defence"", *San Francisco Chronicle*, 23 November 2003, D1.

Conclusion

The media that had been so captivated by Feingold's story in 1973 had become disenchanted by the time of Feingold's death in 1982. There was a sense of disappointment in some stories, for instance, when certain clinical trials found little support for Feingold's hypothesis. Joan Beck, reporting on the seemingly negative results of Harley's trial at the University of Wisconsin, stated that his conclusion 'was not a popular finding in the long battles over how to identify and treat children with hyperactivity'.⁷⁹⁸ The findings, Beck continued, meant that parents, teachers, and physicians had 'to rely on such controversial treatments for hyperactive children as methylphenidate (Ritalin), amphetamine, and/or behavior modification programs at home and in school'.⁷⁹⁹ Harley himself echoed such sentiments, commenting that they 'would have liked nothing better than to find that hyperactivity can be cured through diet. But our study did not bear this out.'⁸⁰⁰

Although there continued to be positive stories about the Feingold diet during the early 1980s, the tenor of the reporting became more negative, or at best neutral, during this period. Following Feingold's death on 23 March 1982, the number of stories about the Feingold diet began to diminish as well; although the occasional story surfaced during the latter part of the decade and during the 1990s about new trials of the diet, they were rare and often published in obscure publications such as the *Brown University Child Behavior and Development Letter* or *Tufts University Diet and Nutrition Letter*.⁸⁰¹ Feingold's death helps to explain the waning of interest – no one with his charisma, determination and credentials stepped forth to carry on his cause – but other factors contributed to the phenomenon as well.

⁷⁹⁸ The next chapter demonstrates that the results of Harley's trial were not at all clear-cut, as Harley himself admitted in a letter in response to Beck's article. Beck, 'Another Miracle Diet', C2; J. Preston Harley, 'Diet for Hyperactivity', *Chicago Tribune*, 29 August 1977, C2.

⁷⁹⁹ Beck, 'Another Miracle Diet', C2.

⁸⁰⁰ J. Preston Harley quoted in Rose Dosti, 'Study Refutes Additive-Hyperactivity Link', *Los Angeles Times*, 14 April, 1977, F1, F4, at p. F1.

⁸⁰¹ Anonymous, 'Is Diet Making Your Child Hyper?' *Tufts University Diet & Nutrition Letter* 7 (1989), 5; Gregory K. Fritz, 'Dietary Intervention for Hyperactive Children Supported in New Study', *The Brown University Child Behavior and Development Letter* 5 (1989), 5.

For Feingold, the influence of the food, chemical and pharmaceutical industries was paramount in influencing media coverage of the debate. Feingold believed as early as 1977 that the media was turning against him, and made his concerns clear in a speech to the Newspaper Food Editors and Writers Association in June 1977. Newspapers, he argued, were reporting the findings of trials that tested his theory without critically assessing the trials for bias and, in some cases, were drawing overly negative conclusions from trial results. The explanation for such actions, according to Feingold, was industry manipulation. Feingold highlighted Harley's University of Wisconsin study, contending that, 'since early January 1976, Dr. Harley has presented his data several times around the country, usually followed in almost every instance by unfavorable reports in the press of the ineffectiveness of the K-P [Kaiser-Permanente] diet'.⁸⁰² Feingold proceeded to charge that 'an analysis of the circumstances and data of Dr. Harley's most recent presentation will illustrate how industry, with a scientific façade manipulates the situation to influence the press to report unwittingly, to industry's advantage'.⁸⁰³ The allergist was particularly alarmed by a *Los Angeles Times* story entitled 'Study Refutes Additive-Hyperactivity Link', which emerged out of a press conference the Dairy Council arranged for Harley at their annual nutrition conference.⁸⁰⁴ Along with criticising Harley's study and its conclusions, Feingold stated: 'I do not know who was responsible for this headline, but it is not only inconsistent with the facts but even with Dr. Harley's written text'.⁸⁰⁵ Moreover, Feingold contended that at 'no time during Dr. Harley's press briefing or in the subsequent articles was there any mention of the \$600,000 support to his [Harley's] Food Research Institute from industry'.⁸⁰⁶

⁸⁰² Feingold, 'A View from the Other Side'.

⁸⁰³ Ibid.

⁸⁰⁴ Dosti, 'Study Refutes'.

⁸⁰⁵ Feingold, 'A View from the Other Side'. Harley himself wrote a letter to the *Chicago Tribune* complaining about how they had misrepresented his results. J. Preston Harley, 'Diet for Hyperactivity', C2.

⁸⁰⁶ Feingold, 'A View from the Other Side'.

The level of industry interference in media stories is difficult to gauge. Although journalists were often remiss in elucidating how specific trials were funded or the connections between investigators and various industries, many did mention that the Nutrition Foundation, for example, was a food, chemical and pharmaceutical industry lobby group. Moreover, most stories represented both sides of the debate, including the story in the *Los Angeles Times* that Feingold criticised. Other factors, therefore, need to be considered in determining why media interest in the Feingold diet petered out.

One factor was that no resolution to the debate appeared to be on the horizon and, as such, journalists, and possibly readers, were tiring of a story which had been running for a decade. In early 1982, for example, reports on the Feingold diet were issued by both a NIH Consensus Development Conference, whose members included the ailing Feingold as well as his critics, and the American Council on Science and Health (ACSH), a non-profit consumer education group founded by Elizabeth Whalen and Frederick Stare and often accused of being a front for the chemical industry.⁸⁰⁷ While the ACSH unsurprisingly concluded ‘that artificial food colors and flavors are not significant causes of hyperactivity’, the NIH group was more circumspect.⁸⁰⁸ Addressing a wide range of issues involved in testing the Feingold diet, the panel concluded that although ‘defined diets should not be universally used in the treatment of childhood hyperactivity at this time ... initiation of a trial of dietary treatment or continuation of a diet in patients whose families and physicians perceive benefits, may be

⁸⁰⁷ According to historians David Rosner and Gerald Markowitz, amongst others, the ACSH has received up to forty per cent of its funding from the food, chemical and pharmaceutical industries, including companies such as American Cyanamid, Dow, Exxon, Monsanto and Union Carbide. David Rosner and Gerald Markowitz, ‘Industry Challenges to the Principle of Prevention in Public Health: The Precautionary Principle in Historical Perspective’, *Public Health Reports* 117 (2002), 508-9. Another dispute is ongoing between the ACSH and CSPI over which organisation truly represents the consumers in matters of science and public health. American Council on Science and Health, ‘CSPI vs. ACSH’, www.acsh.org/about/pageID.86/default.asp, accessed 22 October 2008; Center for Science in the Public Interest, ‘Non-Profit Organizations Receiving Corporate Funding’, www.cspinet.org/integrity/nonprofits/american_council_on_science_and_health.html, accessed 22 October 2008.

⁸⁰⁸ Al Rossiter, Jr., ‘Does Diet Affect Rambunctious Behavior?’, United Press International, 13 January 1982.

warranted'.⁸⁰⁹ In other words, the Consensus Development Conference failed to state definitively that the Feingold diet was efficacious or not and, recognising this, suggested that more research be done to test such theories. Despite the NIH's ambiguity, journalistic interpretations of its statement varied widely: while a *Washington Post* headline read 'Additive-Free Diet Found Not to Curb Hyperactivity', the United Press International newswire read 'Special Diet May Benefit Hyperactive Children'.⁸¹⁰ With no end to the controversy in sight, and no agreement amongst either physicians or journalists on how to weigh the available evidence, it was understandable that the media flocked to other stories following Feingold's death.

Moreover, following 1980, public concern about food additives generally was waning, meaning that specific stories, such as Feingold's, generated less interest than before.⁸¹¹ As journalist Nancy Jenkins observed in 1984, 'the whole food movement gathered strength for a while, but in the late 70s it seemed to have gone underground, along with the rest of what we used to call the counterculture'.⁸¹² Although Jenkins believed that interest in health food was waxing once again, it was 'a national interest, sometimes verging on obsession, with good health and preventative medicine and the role of diet in both', that was spurring the trend, not broader ecological, political and spiritual concerns. As Belasco and Levenstein have emphasised, the yuppies of the 1980s who were targeted by mainstream food companies as a market for healthy food were not so concerned with additive-free food as with low-fat options, as dieting and thinness became entrenched, not for the first or last time, in American

⁸⁰⁹ National Institutes of Health, 'Defined Diets and Childhood Hyperactivity,' *NIH Consensus Statement Online* 4 (13-15 January 1982), www.consensus.nih.gov/1982/1982DietHyperactivity032html.htm, accessed 22 October 2008.

⁸¹⁰ Cristine Russell, 'Additive-Free Diet Found Not to Curb Hyperactivity', *Washington Post*, 15 January 1982, A2; Al Rossiter, Jr., 'Special Diet May Benefit Hyperactive Children', United Press International, 15 January 1982.

⁸¹¹ An exception to this was the controversy over the use of artificial sweeteners, such as saccharin, in soft drinks. Anonymous, 'Key Scientist Favors Elimination of Saccharin Use Within 3 Years', *New York Times*, 12 April 1979, B8; Anonymous, 'House Passes a Delay of Ban on Saccharin', *New York Times*, 25 July 1979, A12; Anonymous, 'Saccharin Held Free of Risk', *New York Times*, 8 November 1985, D20.

⁸¹² Nancy Jenkins, 'Health Food and the Change in Eating Habits', *New York Times*, 4 April 1984, C1.

culture.⁸¹³ Also suggestive of this trend was an article in the ‘Beauty’ section of the *New York Times* which discussed how wealthy Americans were increasingly hiring nutritionists to stay slim.⁸¹⁴ Unlike previous food fads, establishment nutritionists such as Frederick Stare were fully supportive of this ‘fat-phobia’ and, as Levenstein has suggested, so-called ‘Negative Nutrition ... opened new windows of commercial opportunity’ for food manufacturers in the form of Diet Coke, Stouffer’s Lean Cuisine and Weight-Watchers’ products.⁸¹⁵

Concurrently with the decline in stories about the Feingold diet, the tenor of articles and editorials concerning food additives also grew more critical of measures, particularly the Delaney Clause, intended to protect consumers.⁸¹⁶ As a 1982 story by journalist Philip M. Boffey indicated, even concern about the environmental causes of cancer, including food additives and pesticides, had faded, although when food additives did make the news during the 1980s, it was usually because of their potential to cause cancer.⁸¹⁷ The shift mirrored the ebbing of many of the ideals of the 1960s, and was made manifest in the election of right wing Republican Ronald Reagan (1911-2004) in 1980. As Levenstein describes, ‘lust for wealth displaced older ideas of public service in Washington, drove considerations of responsibility to clients, stockholders, and the public from Wall Street boardrooms’.⁸¹⁸ Such a philosophy was reflected in the administration’s support for the food, chemical and pharmaceutical industries and successful attempts to deregulate such industries.⁸¹⁹

⁸¹³ Belasco, *Appetite for Change*, 1-3; Levenstein, *Paradox of Plenty*, 237-41.

⁸¹⁴ Alexandra Penney, ‘The Year of the Nutritionist’, *New York Times*, 1 July 1979, SM8.

⁸¹⁵ Levenstein, *Paradox of Plenty*, 241-3.

⁸¹⁶ Anonymous, ‘The Bitter Verdict Against Saccharin’, *New York Times*, 11 March 1977, 16; Anonymous, ‘The Case of the Useful Carcinogen’, *New York Times*, 7 December 1978, A22.

⁸¹⁷ Philip M. Boffey, ‘Cancer Experts Lean Towards Steady Vigilance, but Less Alarm, on Environment’, *New York Times*, 2 March 1982, C1-C2.

⁸¹⁸ Levenstein, *Paradox of Plenty*, 236.

⁸¹⁹ Karen De Witt, ‘Challenging the Additive Rule’, *New York Times*, 10 December 1980, C1, C13, at p. C1; Karen De Witt, ‘Just a Regulated Day in the Life of an Ordinary Citizen’, *New York Times*, 12 April 1981, E9.

Despite the decline in media interest, however, researchers continued to test the diet during the 1980s, albeit intermittently, and parents continued to learn about the diet through other means. Debate about Feingold's theory continued to simmer, but it did not attract the attention of the mainstream media. Although the media played a major role in publicising the Feingold diet to millions of Americans, its overall impact on how Americans assessed Feingold's hypothesis is more difficult to determine. Depending on which story an individual happened to read, they might read a glowing endorsement, a scathing indictment or a confounding account of a protracted debate, any of which might or might not have accorded with what they were predisposed to think about food additives, hyperactivity or the role of the state in regulating industry. In the case of the Feingold diet, therefore, the media as a whole did not influence public opinion as much as it reflected the complexities involved in attempting to determine whether Feingold's theory was valid or not.

Although discussion of the Feingold diet in the media did little to resolve the debates regarding his theory, it did encourage scientists to examine his hypothesis more closely. Demanding proof, demonstrated in double-blind clinical trials, that food additives were a causative factor in hyperactivity, medical researchers began testing Feingold's theory soon after the allergist's 1973 AMA conference and published their findings in leading medical journals. The following chapter analyses these trials, including how they were designed, conducted, interpreted and used by those testing Feingold's theory, and also examines more broadly how physicians answered for themselves, and by extension most of the general community, questions about the Feingold diet.

Chapter 9

Testing the Feingold Diet

During the period between the publication of *Why Your Child is Hyperactive* in 1974 and Feingold's death in 1982, researchers in the United States, Canada and Australia designed dozens of trials that tested Feingold's theory.⁸²⁰ The prevailing opinion that emerged from these trials, reflected in summaries of the trials and reviews of alternative treatments for hyperactivity, was that the Feingold diet did not stand up to scientific scrutiny and that parents of hyperactive children should consider other treatment options.⁸²¹ In contrast, FAUS and groups such as CSPI argued that tests of the Feingold diet provided solid evidence in support of Feingold's hypothesis.⁸²² It is somewhat understandable that certain parties, for example, the Nutrition Foundation on one hand and FAUS on the other, would interpret the test results in manners conducive to their own vested interests, but this leaves unanswered the question of what the tests of the Feingold diet did in fact reveal about its validity.

⁸²⁰ British researchers were relatively slow to investigate the Feingold diet, although they have led research into the hypothesis since the mid-1980s. This is partly because the concept of hyperactivity as a discrete childhood behaviour disorder did not emerge in Britain until the 1980s, twenty-five years after it had become predominant in the United States. According to British criminologist Steven Box (1937-1987), although some prominent child psychiatrists, notably Sir Michael Rutter (b. 1933), used the term 'hyperkinesis' during the 1970s, the British Education of Education and Science described children who could have been diagnosed as hyperactive as being either 'maladjusted' or 'medium educational subnormal'. More discussion on how Feingold's hypothesis was received in different countries can be found in Chapter 11. Steven Box, 'Preface', in Peter Schrag and Diane Divoky, *The Myth of the Hyperactive Child: And Other Means of Child Control* (New York: Penguin Books, [1975] 1982), 7-30, at p. 17; Michael Rutter, J. Tizard, W. Yule, P. Graham and K. Whitmore, 'Research Report: Isle of Wight Studies, 1964-1974', *Psychological Medicine* 6 (1976), 313-32; Seija T. Sandberg, Michael Rutter and E. Taylor, 'Hyperkinetic Disorder in Psychiatric Clinic Attenders', *Developmental Medicine and Child Neurology* 20 (1978), 279-99.

⁸²¹ The opinion that the trials conducted during the 1970s and early 1980s disproved the Feingold diet was expressed in a number of review articles, for example, Gerald S. Golden, 'Nonstandard Therapies in the Developmental Disabilities', *American Journal of Diseases of Children* 134 (1980), 487-91, at p. 489; J. A. Mattes and R. Gittelman, 'Effects of Artificial Food Colorings in Children with Hyperactive Symptoms' *Archives of General Psychiatry* 38 (1981), 714-18; Kenneth A. Kavale and Steven R. Forness, 'Hyperactivity and Diet Treatment: A Meta-Analysis of the Feingold Hypothesis', *Journal of Learning Disabilities* 16 (1983), 324-330; Jeffrey A. Mattes, 'The Feingold Diet: A Current Reappraisal', *Journal of Learning Disabilities* 16 (1983), 319-23; Esther H. Wender, 'The Food Additive-Free Diet in the Treatment of Behavior Disorders: A Review', *Journal of Developmental and Behavioral Pediatrics* 7 (1986), 35-42; Steer, 'Managing Attention Deficit/Hyperactivity Disorder', i22.

⁸²² Michael F. Jacobson and David Schardt, *Diet, ADHD and Behavior: A Quarter Century Review* (Washington, D.C.: Center for Science in the Public Interest, 1999); Feingold Association of the United States, 'Diet and ADHD', www.feingold.org/pg-research.html accessed 16 December 2008, accessed 8 April 2009.

Western society's faith in the power of scientific knowledge suggests that double-blind clinical trials of the Feingold diet should have unequivocally demonstrated whether or not Feingold's idea was tenable.⁸²³ Close examination of the tests of the Feingold diet, however, reveals that the trials were anything but conclusive. Although there were undoubtedly some trials that yielded negative results, there were others that were decidedly positive. Indeed, what emerges from historical analysis of the dozens of tests of Feingold's theory are not definitive answers about the efficacy of the Feingold diet, but instead more questions about how researchers designed, conducted and most importantly, interpreted the trial results. The inconclusivity of the tests not only suggests that other, non-scientific, factors were more influential in shaping the opinions of various parties regarding the Feingold diet, but also raises questions about the effectiveness of double-blind clinical trials in resolving similar debates, particularly those in the fields of psychiatry, nutrition and allergy.

It is clear from reviews of the trials that, while Feingold's detractors were liable to ignore positive results, his supporters were inclined to downplay negative results.⁸²⁴ Moreover, the trials themselves often contained methodological problems, making it difficult to understand how the results of such trials were perceived as being conclusive. Researchers differed considerably with respect to how to interpret their own results and, therefore, whether or not their results should be counted as being supportive or critical of the Feingold diet. While some researchers were unimpressed if large percentages of their sample reacted to food

⁸²³ As sociologists Harry Collins and Trevor Pinch explain, randomised controlled trials, of which double-blind trials are the most common and most accurate type, are regarded as the 'gold standard for scientific medicine'. Although such trials are seen to be the height of scientific research, Collins and Pinch maintain that this is only the case because medical knowledge is unable to cope with the power of the placebo effect. As this chapter demonstrates, controlling for placebo was of utmost importance in the trials of the Feingold diet. Harry Collins and Trevor Pinch, *Dr. Golem: How to Think About Medicine* (Chicago: University of Chicago Press, 2005), 32-4.

⁸²⁴ This tendency was recognised in a more recent review of the Feingold diet research literature. David W. Schab and Nhi-Ha T. Trinh, 'Do Artificial Food Colors Promote Hyperactivity in Children with Hyperactive Syndromes? A Meta-Analysis of Double-Blind Placebo-Controlled Trials', *Journal of Developmental & Behavioral Pediatrics* 25 (2004), 423-34.

additives, other researchers were alarmed if only a few of their sample responded strongly. Finally, although Feingold's detractors consistently claimed that trial results were the basis of their critique, they also attacked the diet using arguments that had nothing to do with the trials, contending, for example, that the diet was an impractical intervention for most American families. Given all of these factors, therefore, it would be difficult for any physician, parent or policy maker to determine whether the Feingold diet worked or not.

The beginning of this chapter provides a close analysis of the trials designed to test the Feingold diet. It considers the role of the Nutrition Foundation in influencing how Feingold's theory would be assessed and outlines the methodological problems that undermined many of the trials. It then proceeds to examine how the results of the trials were interpreted by both the researchers who conducted them and outside observers. The reasons why researchers could come to significantly different conclusions about a particular trial are also discussed. Finally, the chapter concludes by demonstrating that, while most researchers quickly developed fixed opinions about Feingold's hypothesis, there were others who fluctuated with respect to whether or not it was valid. This raises questions not only about how and why scientists make decisions about controversial issues, but also about the effectiveness of double-blind trials in helping them resolve such debates.

What to Test, How to Test, Who to Test: Methodological Difficulties

Following Feingold's presentations to the AMA in 1973 and 1974, five California-based studies were undertaken at medical centres and schools to test his idea.⁸²⁵ Two studies were also carried out in Australia and published in the *Medical Journal of Australia*, prompting Feingold to visit the country on a lecturing tour in September 1976.⁸²⁶ None of the studies were controlled, however, and two of those conducted in California were carried out at Kaiser Permanente clinics with the involvement of Feingold himself. The reports emanating from these clinical studies were generally positive and attracted the attention of the media and the medical community.⁸²⁷ Concerned that no controlled studies had been conducted to assess the validity of Feingold's theory, both the FDA and the Nutrition Foundation recommended in 1975 that controlled double-blind trials be designed to test it.⁸²⁸ Early in that year the Nutrition Foundation recruited over a dozen physicians, nutrition scientists and psychologists to form the National Advisory Committee on Hyperkinesis and Food Additives (NACHFA) to review the trials and make judgments on their findings.⁸²⁹

In setting out the rationale for their investigation of Feingold's hypothesis, NACHFA emphasised their suspicion that the benefits of the Feingold diet were due to placebo, rather

⁸²⁵ C. Keith Conners, *Food Additives and Hyperactive Children* (New York: Plenum Press, 1980), 9-10.

⁸²⁶ Peter S. Cook and Joan M. Woodhill, 'The Feingold Dietary Treatment of the Hyperkinetic Syndrome', *Medical Journal of Australia* 2 (1976), 85-9; Louis K. Salzman, 'Allergy Testing, Psychological Assessment and Dietary Treatment of the Hyperactive Child Syndrome', *Medical Journal of Australia* 2 (1976), 248-51. The Australian studies generated a considerable response, as did those reported in the United States. Although most of the letters to the *Medical Journal of Australia* concerning the studies and the Feingold diet were positive, New Zealander psychiatrist John Werry's editorial (discussed below), in contrast, was a scathing attack on Feingold's methods and motives. Australian medical interest in the risks chemical exposures posed to human health was spurred in part by immunologist Stephen Boyden's 1972 speech to the Australian Medical Congress, in which he warned about the "chemicalization" of the environment'. In particular, Boyden claimed that 'the first symptoms of exposure to many toxic chemicals are not physiological, but psychological, and include such symptoms as confusion, personality changes, fatigue, loss of memory and mental dullness'. Peter Cook and Joan Woodhill, a child psychiatrist and nutritionist team, cited Boyden's concerns in their paper on the Feingold diet. Stephen Boyden, 'The Environment and Human Health', *Medical Journal of Australia* 1 (1972), 1229-34, at p. 1231; John S. Werry, 'Food Additives and Hyperactivity', *Medical Journal of Australia* 2 (1976), 281-2.

⁸²⁷ Conners, *Food Additives and Hyperactive Children*, 10.

⁸²⁸ Helen Tryphonas and Ronald Trites, 'Diet and Hyperactivity', *Nutrition Bulletin* 9 (1984), 24-31, at pp. 27-8.

⁸²⁹ NACHFA, *Report to the Nutrition Foundation* (New York: Nutrition Foundation, 1975).

than the elimination of food additives. Placebo, they contended, could operate in three separate ways in both clinical observations and uncontrolled trials. First, dietary changes affecting the entire family could cause ‘alterations in family dynamics ... related to the reported improvement in the child’. Secondly, Feingold’s charisma and confidence about his regimen could generate positive expectations in the patients and their families, thus affecting parental perceptions of improvement. Thirdly, ‘parents and teachers who rate the children know that they are on the diet and this knowledge may influence their ratings’.⁸³⁰ Given the many ways in which placebo could influence trial results, and the ‘enormous expenditure’ inherent in ‘producing a wide variety of dietary products in identical pairs containing, or free of, specific chemical ingredients’, NACHFA recommended the use of challenge studies whereby a single food, containing a particular food additive, would be randomly served to participants in order to determine if it triggered increased hyperactivity.⁸³¹ Although they warned that other factors, including the compliance of participants, the validity of behavioural observations and the large number of substances eliminated in the Feingold diet, complicated interpretation of the trials, NACHFA maintained that ‘data from critically designed and executed studies, free of the deficiencies noted, must be available before firm conclusions can be reached on the Feingold hypothesis’.⁸³²

Despite the call for the Feingold diet to be tested in controlled double-blind trials, and the guidelines set out by NACHFA to design such trials, methodological problems plagued nearly all of the trials conducted during the 1970s and early 1980s and, according to some researchers, discouraged others from testing the theory. Indeed, attempting to address the

⁸³⁰ NACHFA, *Final Report to the Nutrition Foundation* (New York: The Nutrition Foundation, 1980), Appendix, vi. Or, as described more concisely in a 1977 letter to the *Medical Journal of Australia* which described the Feingold diet as a ‘very good placebo’: ‘Many children described as hyperactive are in fact responding to their own parents’ anxiety, which is alleviated at least temporarily by treatment which appears both complicated and powerful.’ J. C. M. Friend, ‘The Syndrome of Childhood Hyperactivity’ *Medical Journal of Australia* 1 (1977), 819-23, at p. 822.

⁸³¹ NACHFA, *Final Report to the Nutrition Foundation*, Appendix, vii-viii.

⁸³² *Ibid.*, vii.

methodological weaknesses of uncontrolled trials merely led to different methodological problems. Writing in 1987, a team led by psychiatrist Mortimer D. Gross stated:

a major reason for the dearth of controlled studies is the difficulty in performing them when food is involved: 1) unless the subjects are confined to a strictly controlled environment, cheating is all too easy; 2) children are difficult to persuade to stick to a prescribed diet...3) ideally the food being tested should be disguised so that the subjects are blind to what they are ingesting - and this is difficult to manage; and 4) the raters should be blind to what subjects are eating, and this, too, is difficult to arrange.⁸³³

At the heart of the methodological problems was the general perception that hyperactivity was a complex condition influenced by many factors. Although most physicians during the mid 1970s believed that the disorder was chiefly a neurological condition, NACHFA's concerns about placebo implied that a child's social, domestic and educational environment also played a role in at least exacerbating hyperactivity. In order to establish a clear, definitive link between food additives and hyperactivity, all such factors had to be controlled. Moreover, discrepancies existed regarding how to identify the disorder and all of its constituent parts, including not only hyperactive behaviour, but also distractibility, impulsivity, defiance and aggression. Although Conners' parent and teacher questionnaires, designed during the 1960s by psychologist C. Keith Conners, were used in many of the Feingold trials, there was still an element of subjectivity on behalf of the person observing; what was pathological, disordered behaviour to one parent or teacher, for example, could be energetic play to another. As an anonymous editorial for the *Lancet* explained in 1979, Feingold's 'hypothesis would be difficult to test even if the state of hyperactivity in children

⁸³³ Mortimer D. Gross, Ruth A. Tofanelli, Sharyl M. Butzirus and Earl W. Snodgrass, 'The Effects of Diets Rich in and Free from Additives on the Behaviour of Children with Hyperkinetic and Learning Disorder', *Journal of the American Academy of Child and Adolescent Psychiatry* 26 (1987), 53-5, at p. 53. Ironically, FAUS has described the trial conducted by Gross, *et al* as one of the most poorly-designed tests of the diet, listing a dozen methodological problems. FAUS, 'Diet and ADHD', www.feingold.org/pg-research.html, accessed 21 January 2009.

were a precise and readily recognisable entity. It is not ... hyperactivity remains a clinical concept of doubtful validity.⁸³⁴

If hyperactivity was in itself an illusive concept to define, identify and assess, the implications of Feingold's hypothesis made comprehensive testing of the Feingold diet even more problematic. Feingold claimed that there were thousands of additives in the food supply that could trigger hyperactivity, and that certain salicylate-laden fruits and vegetables could also invoke reactions. Moreover, not all children reacted to the same chemicals.⁸³⁵ Since testing thousands of substances individually was logistically and economically impossible, many researchers limited their inquiry to a single chemical, such as the food dye tartrazine yellow, or a combination of common food dyes.⁸³⁶

The issue of exactly what to test was a source of contention between Feingold, his supporters and NACHFA. Although Feingold emphasised the sheer number of potentially problematic chemicals in the food supply, when he met with NACHFA in 1975 to discuss how to test his hypothesis, he 'recommended that, in view of the complexity of the problem and the many compounds involved, studies be designed focusing on the limited list of colors, which lend themselves to better control'.⁸³⁷ NACHFA, therefore, advocated that artificial flavours, as well as the salicylate-laden fruits and vegetables, food preservatives and other food additives, not be tested, leaving food colours as the sole substance of interest. The advisory committee also argued that there were other reasons for omitting the other substances, stating that 'the

⁸³⁴ Anonymous, 'Feingold's Regimen for Hyperkinesis', *Lancet* 2 (1979), 617-8, at p. 617.

⁸³⁵ Indeed, this is reflected in the oral history interviews of Feingold families – see Chapter 10.

⁸³⁶ F. Levy, S. Dumbrell, G. Hobbes, M. Ryan, N. Wilton, J. M. Woodhill, 'Hyperkinesis and Diet: A Double-Blind Crossover Trial with a Tartrazine Challenge', *Medical Journal of Australia* 1 (1978), 61-4; David, 'Reactions to Dietary Tartrazine'.

⁸³⁷ Benjamin F. Feingold quoted in Bernard Rimland, 'The Feingold Diet: An Assessment of the Reviews by Mattes, by Kavale and Forness and Others', *Journal of Learning Disabilities* 16 (1983), 331-3, at p. 331. The limited list of colours included nine dyes that were approved for use in the US and nine that were approved in Canada. The lists were slightly different, since Red #2 was banned in the US and allowed in Canada, the reverse being true for Red #40. NACHFA, *Final Report to the Nutrition Foundation*, 9.

chemical components of synthetic food flavorings are usually identical to the chemicals contained in natural foods’, and that ‘such a challenge substance would have to be prepared from a list of over a thousand chemicals, and it would be impossible to disguise flavoring in the placebo food’.⁸³⁸ Another problem, not cited by NACHFA but elsewhere, was that there were no government guidelines on the average amount of flavourings consumed, so researchers had little idea about what dosages to test.⁸³⁹ As a result, the challenge studies that NACHFA recommended were only expected to test food dyes, and the Nutrition Foundation proceeded to create a placebo cookie that contained all nine of the dyes approved by the Food, Drug and Cosmetics Act of 1938 in direct proportion to the volume of each dye sold in the United States.⁸⁴⁰ These cookies were supplied by the Nutrition Foundation to researchers who applied to them for funding, having ‘submitted protocols containing appropriate scientific safeguards to assure the double-blind nature of their observations’.⁸⁴¹

The focus on food dyes was not what Feingold had intended; food dyes were supposed to be the starting point of a series of tests on all types of additives, rather than the only substance tested. The focus on testing dyes at the expense of other food additives, according to Feingold, meant that his hypothesis was not being fully tested and incorrectly suggested that he thought dyes were the most important factor in triggering hyperactivity. As one of Feingold’s supporters, psychologist and autism researcher Bernard Rimland (1928-2006),

⁸³⁸ NACHFA, *Final Report to the Nutrition Foundation*, 7-9.

⁸³⁹ Bonnie J. Kaplan, ‘The Relevance of Food for Children’s Cognitive and Behavioural Health’, *Canadian Journal of Behavioural Science* 20 (1988), 359-73.

⁸⁴⁰ Both the challenge and the placebo cookies designed by the Nutrition Foundation used chocolate in order to disguise the amount of dye in the challenge cookies. Although not questioned at the time, psychologist Bonnie Kaplan, writing in 1988, stated that both chocolate and sugar were not longer thought to be suitable substances for a placebo food. Kaplan, ‘The Relevance of Food’, 360.

⁸⁴¹ For example, C. H. Goyette, C. K. Conners, T. A. Petti and L. E. Curtis ‘Effects of Artificial Colors on Hyperkinetic Children: A Double-Blind Challenge Study’, *Psychopharmacological Bulletin* 14 (1978), 39-40; J. P. Harley, C. G. Matthews, P. Eichman, ‘Synthetic Food Colors and Hyperactivity in Children: A Double-Blind Challenge Experiment’, *Pediatrics* 62 (1978), 975-83, at p. 976; J. Ivan Williams, Douglas M. Cram, Frances T. Tausig, and Evelyn Webster, ‘Relative Effects of Drugs and Diet on Hyperactive Behaviors: An Experimental Study’, *Pediatrics* 61 (1978), 811-17, at p. 812; NACHFA, *Final Report to the Nutrition Foundation*, 2, 9-10.

exclaimed: ‘How researchers can claim they have tested “the Feingold diet,” which eliminates over 3,000 additives, by conducting experiments based on fewer than 10 dyes, is beyond me.’⁸⁴²

On one level, such complaints were somewhat disingenuous. Feingold had recommended that testing synthetic dyes was the best placebo to start and colours were likely the most iconic and feared food additive. If coal-tar-based food dyes such as Brilliant Blue and Sunset Yellow could not be conclusively found to trigger hyperactivity, Feingold’s hypothesis was certainly in doubt, if not theoretically, then at least in the eyes of the medical and lay community. But even if testing synthetic dyes was the key task in testing the Feingold diet, then there were still other methodological problems that made the results of dye-based trials difficult to interpret.

One important aspect, for example, was the amount of dye to be tested, or dosage level. The amount of dye in the NACHFA cookies, for example, was based upon a calculation of the average daily per capita consumption of food dyes in the US in the years 1973 and 1974.

The advisory committee admitted, however, that following:

the first two or three challenge studies, concern was expressed that the dose of food coloring employed may be much less than the amount of coloring consumed by children. It was argued by some that children, on the average, consume a much higher proportion of artificially colored foods than do adults.⁸⁴³

Or, as Rimland put it:

The dosage levels were ridiculously small. Even if one were to accept the wholly unwarranted conclusion that seven to 10 food colorings were the overwhelming important factor in the Feingold diet, one would still have to reject the bulk of the studies, since the researchers used almost trivially small doses of colorings.⁸⁴⁴

⁸⁴² Rimland, ‘The Feingold Diet’, 331.

⁸⁴³ NACHFA, *Final Report to the Nutrition Foundation*, 10.

⁸⁴⁴ Rimland, ‘The Feingold Diet’, 331.

Rimland's hyperbole notwithstanding, there was a wide range in the doses of food dyes used by researchers, spanning between 1.2 mg to 150 mg.⁸⁴⁵ The cookies designed by the Nutrition Foundation, and used by many researchers, contained 13 mg of dye, but since two cookies were intended to be consumed each day by each child in the trials, the total amount of dye was 26 mg. This amount was calculated by adding up the entire amount of dye certified by the FDA per year and dividing it by the American population and the number of days in the year, a decidedly rough approximation. Nevertheless, NACHFA soon recognised that this was lower than the average consumed by children, and created a 'soda-pop drink' that contained 36 mg for a subsequent trial.⁸⁴⁶ But this amount was also below the FDA average of 57.5 mg, and far below FDA estimates of what children at the high end of the spectrum might consume, namely, as much 121 mg for children in the 90th percentile and rising up to a maximum of 315 mg per day.⁸⁴⁷ When combined with the fact that the amount of each of the nine dyes in the cookie was proportionate to the dye's relative use in the food supply (for example, Blue # 2 only accounted for 1.7% of dyes in the food supply, so it only made up 1.7% of the dyes in the cookie), participating children could be getting minimal amounts of dye to which they might be reactive.⁸⁴⁸

⁸⁴⁵ Strangely, both the studies using the lowest dose and the highest dose yielded positive results. Also interesting is the fact that two of the most recent studies, both of which also yielded positive results, used relatively low dosages of between 20 and 25 mg. Terry L. Rose, 'The Functional Relationship between Artificial Food Colors and Hyperactivity', *Journal of Applied Behavior Analysis* 11 (1978), 439-46, at p. 441; James W. Swanson and Marcel Kinsbourne, 'Food Dyes Impair Performance of Hyperactive Children on a Laboratory Learning Test', *Science* 207 (1980) 1485-7; B. Bateman, J. O. Warner, E. Hutchinson, T. Dean, P. Rowlandson, C. Grant, J. Grundy, C. Fitzgerald and J. Stephenson, 'The Effects of a Double-Blind, Placebo Controlled, Artificial Food Colourings and Benzoate Preservative Challenge on Hyperactivity in a General Population Sample of Preschool Children', *Archives of Disease in Childhood* 89 (2004), 506-11, at p. 507; Donna McCann, Angelina Barrett, Alison Cooper, Debbie Crumpler, Lindy Dalen, Kate Grimshaw, Elizabeth Kitchin, Kris Lok, Lucy Porteous, Emily Prince, Edmund Sonuga-Barke, John O. Warner, Jim Stevenson, 'Food Additives and Hyperactive Behaviour in 3-Year-Old and 8/9-Year-Old Children in the Community: A Randomised, Double-Blinded, Placebo-Controlled Trial', *Lancet* 370 (2007), 1560-7, at p. 1561.

⁸⁴⁶ NACHFA, *Final Report to the Nutrition Foundation*, 10.

⁸⁴⁷ T. J. Sobotka, 'Estimates of Average, 90th Percentile and Maximum Daily Intakes of FD & C Artificial Food Colors in One Day's Diets Among Two Age Groups of Children', Food And Drug Administration Memorandum, July 1976; Connors, *Food Additives and Hyperactive Children*, 105; Alexander G. Schauss, 'Nutrition and Behaviour: Complex Interdisciplinary Research', *Nutrition and Behavior* 3 (1984), 23.

⁸⁴⁸ NACHFA, *Final Report to the Nutrition Foundation*, 10.

Even reviews of the Feingold trials that dismissed the Feingold diet admitted that it 'is conceivable that previous studies ... used inadequate doses of food colourings'.⁸⁴⁹ Another researcher admitted that 'the doses employed by us, and most of our fellow investigators, are 50 times less than the maximum allowable daily intakes (ADI's) recommended by the Food and Drug Administration'.⁸⁵⁰ Facing concern about the amount of dye used in trials, NACHFA claimed that there was 'a technical limitation to the amount of food coloring that can be incorporated into a food without coloring the mouth and fingers ... and thus preventing the disguise of the placebo challenge'.⁸⁵¹ Despite this hindrance, which could have been overcome with some ingenuity, a number of researchers opted for higher levels of dye and reported results in favour of Feingold's hypothesis.⁸⁵²

The issue of challenge materials hampered trials in other ways. In one trial, researchers expected the children to eat six challenge cookies per day. This amount proved to be too much for one child's appetite, and too much for two parents who were alarmed by the reactions their children had after consuming what they thought was the challenge cookie. Although one of the parents correctly guessed that her child was consuming the challenge cookie, the other child was consuming the placebo.⁸⁵³ A similar situation occurred in one of Connors' trials; a mother took her son out of the trial when his behaviour deteriorated rapidly following the ingestion of a cookie, but it turned out that the cookie was a placebo.⁸⁵⁴ In

⁸⁴⁹ Mattes and Gittelman, 'Effects of Artificial Food Colorings', 715.

⁸⁵⁰ Bernard Weiss, J. Hicks Williams, Sheldon Margen, Barbara Abrams, Bette Caan, L. Jay Citron, Christopher Cox, Jane McKibben, Dale Ogar and Stephen Schultz, 'Behavioural Responses to Artificial Food Colours', *Science* 207 (1980), 1487-9.

⁸⁵¹ NACHFA, *Final Report to the Nutrition Foundation*, 11.

⁸⁵² For example, in the Swanson and Kinsbourne study, the dye was disguised in a capsule, as was the placebo. Swanson and Kinsbourne, 'Food Dyes Impair Performance'; J. Egger, C. M. Carter, P. J. Graham, D. Gumley and J. F. Soothill, 'Controlled Trial of Oligoantigenic Treatment in the Hyperkinetic Syndrome', *Lancet* 325 (1985), 540-5, at p. 540.

⁸⁵³ Mattes and Gittelman, 'Effects of Artificial Food Colorings', 717.

⁸⁵⁴ C. Keith Connors, *Feeding the Brain: How Foods Affect Children* (New York: Plenum Press, 1989), 12.

other trials, including more recent ones, parents were wary of subjecting their children to the challenge foods and removed their children from the study.⁸⁵⁵

Another methodological problem associated with testing the Feingold diet was the compliance of the children who participated in trials. For example, pre-school-aged children in J. Preston Harley's study at the University of Wisconsin made, on average, 1.33 dietary infractions per week. Although Harley believed this to be a high rate of compliance, Feingold claimed that a single dietary infraction could affect a hyperactive child adversely for up to six days, thus compromising the results of Harley's trial.⁸⁵⁶ In order to minimise infractions, one trial was held at a hospital and another at an Illinois summer camp for children with learning disorders.⁸⁵⁷ The children at the camp were fed the Feingold diet during one week and then returned to an additive-rich diet the following week. Although they were better able to control what the children ate, other factors complicated how the investigators interpreted whether or not their behaviour had improved:

One result was unmistakable: the children were not happy with the Feingold diet. The teachers had the feeling that there would have been a rebellion had it lasted longer than a week. They particularly disliked the colourlessness of the food, and missed the mustard and ketchup. ... The strict Feingold diet appears to be distasteful to be the typical American child.⁸⁵⁸

Given the rebellious attitude of the children at the camp, who were predominantly teenagers, not young children, the interpretation of behaviour, which was not observed firsthand but via videotapes at four minute intervals, was problematic. The summer camp study had other methodological problems as well. For instance, only 19 of the 39 children studied during the

⁸⁵⁵ Bateman, *et al*, 'Effects of a Double-Blind', 507; I. Pollock, J.O. Warner, 'Effects of Artificial Food Colours on Childhood Behaviour', *Archives of Disease in Childhood* 65 (1990), 74-7, at p. 76.

⁸⁵⁶ Feingold, 'Hyperkinesis and Learning Disabilities'; J. Preston Harley, Roberta S. Ray, Lawrence Tomasi, Peter L. Eichman, Charles G. Matthews, Raymond Chun, Charles S. Cleeland and Edward Traisman, 'Hyperkinesis and Food Additives: Testing the Feingold Hypothesis' *Pediatrics* 61 (1978), 818-28, at p. 821.

⁸⁵⁷ T. J. David, 'Reactions to Dietary Tartrazine', *Archives of Disease in Childhood* 62, (1987), 119-22; Gross, *et al*, 'The Effects of Diets Rich in and Free from Additives'.

⁸⁵⁸ Gross, *et al*, 'The Effects of Diets Rich in and Free from Additives', 54-5.

comparatively short two-week-long trial had been diagnosed with hyperactivity, and eighteen of those remained on stimulant medication throughout the trial. Moreover, three children, two of them hyperactive, were sent home for various behavioural problems during the second week when additives were re-introduced to the diet. Despite this, and the impressions of the camp director and teachers that the children had behaved worse during the second week, the researchers concluded that the Feingold diet was ineffective.⁸⁵⁹

Other methodological problems made interpretation a difficult task. Most researchers tested small numbers of children, usually ranging between ten and twenty, and not all children managed to complete the full trial period. Given all of the concerns about methodology, small sample sizes were perhaps understandable, as they made controlling many aspects of the trials easier, but then questions could be raised about the statistical significance of such small trials.⁸⁶⁰ Another issue was when to challenge children with additives following a period on an elimination diet. While many researchers waited three to four weeks before introducing the challenges, others believed that testing four to six days after additives were eliminated from the diet was a better strategy, since this was methodologically similar to how allergists tested other food allergies.⁸⁶¹

In some ways, the methodological problems that plagued the trials of the Feingold diet were understandable. Hyperactivity was a diverse syndrome, characterised by many different types of behaviour. Feingold's theory involved thousands of potentially harmful substances, any of which he claimed could trigger hyperactivity in a child. Moreover, children's behaviour was difficult to explain, and a plethora of educational, emotional, neurological,

⁸⁵⁹ *Ibid.*, 53-5.

⁸⁶⁰ Indeed, later trials have involved sample sizes of hundreds of children. For example, Bateman's 2005 trial involved 277 children, whittled down from an initial population of 2878, and McCann's study tested 297 children. Bateman, *et al*, 'Effects of a Double-Blind'; McCann, *et al*, 'Food Additives and Hyperactivity'.

⁸⁶¹ Connors, *Food Additives and Hyperactive Children*, 105-6.

social and familial factors had to be eliminated before proving unequivocally that food additives were at fault. Combining all of these issues with the inherent difficulty of conducting a trial involving food made testing Feingold's hypothesis a complicated and potentially frustrating prospect.

Despite the apparent difficulties in designing trials that would test the Feingold diet, the willingness of researchers to overcome such problems was less clear. When asked why many of the trials were so poorly designed, for instance, toxicologist Bernard Weiss, who conducted a trial himself, answered that many trials:

were carried out by people who had the answers before they did the study. I mean that's the reason I did it. I said wait a minute, the FDA does not screen for neurotoxicity. How come? That's crazy! There were some people within it who thought it was important, but they could never get anybody to listen. So I thought, this is an important toxicology issue. It's not a question of diet and kids, but an issue for regulatory agencies. Why the hell are you not examining food additives for potential neurotoxicity?⁸⁶²

Although most other researchers were unwilling to admit that they had preconceived notions about the validity of Feingold's hypothesis prior to conducting research, Weiss' admission suggests that this is a distinct possibility. Canadian researchers J. Ivan Williams and Douglas M. Cram, for instance, asserted that 'there has been interest in testing [the Feingold hypothesis] if only to disprove it'.⁸⁶³ As described below, the manner in which investigators and reviewers interpreted and discussed Feingold's hypothesis indicate that most people who took the time to debate the Feingold diet had strong ideas about it, and their opinions were not always swayed by the results from double-blind trials.

Interestingly, Weiss also hailed a trial conducted in the late 1980s by psychologist Bonnie Kaplan at the University of Calgary's Alberta Children's Hospital as being 'a terrific clinical

⁸⁶² Bernard Weiss, Interview.

⁸⁶³ Williams and Cram, 'Diet in the Management of Hyperkinesis', 243.

trial’ and ‘a superb study’.⁸⁶⁴ One key difference between Kaplan’s study and most of the others was that she and her associates manipulated the entire nutrient intake of her subjects. This ‘dietary replacement design’ was expensive and difficult to organize, but permitted ‘the evaluation and control of many variables not possible with the more popular challenge designs’.⁸⁶⁵ In other words, instead of testing only a limited quantity of food dyes, as most trials did, Kaplan’s study tested a broader range of food additives in the quantities that they would normally be present in a child’s diet.⁸⁶⁶

The other difference between Kaplan’s study and many others was her stated rationale for conducting research into the Feingold diet. Although she had a longstanding interest in nutrition and behaviour, Kaplan had not heard about Feingold’s hypothesis until 1979 when Jane McNicol, a dietician working at the Children’s Hospital in Calgary, brought it to her attention. McNicol, according to Kaplan, ‘wasn’t a Feingoldian, ... She just believed she really could see improvements in children who ate a healthier diet’.⁸⁶⁷ Kaplan recalled how she ‘looked at the Feingold-simulated research and, frankly, at that time it was pretty poor; ... basically we decided ... why couldn’t we do a better study at Children’s Hospital?’⁸⁶⁸

Explaining her motives for investigating the topic, Kaplan claimed that:

I was about as open-minded as I’ve been about any study I’ve done. I hadn’t seen it affect any children; I had no clinical experience. What I remember about my attitude was, ‘My God, I could do a better job than some of them out there’ ... it does sound arrogant, but I saw it as a challenge in experimental research and design, but I had nothing invested in the outcome.⁸⁶⁹

⁸⁶⁴ Ibid.

⁸⁶⁵ Bonnie J. Kaplan, Jane McNicol, Richard A. Conte, H. K. Moghadam, ‘Dietary Replacement in Preschool-Aged Hyperactive Boys’, *Pediatrics* 83 (1989), 7-17, at p. 7.

⁸⁶⁶ One of the creative ways in which Kaplan attempted to control what the children were eating away from home was to produce t-shirts for the children to wear that read: ‘Don’t feed me, I’m allergic.’ The t-shirts were particularly helpful for children living in apartments.

⁸⁶⁷ Bonnie Kaplan, Telephone Interview, 5 November 2007.

⁸⁶⁸ Ibid.

⁸⁶⁹ Ibid.

In contrast, many of the uncontrolled studies of the Feingold diet were conducted by clinicians who had first employed the diet in their clinic and experienced success with it.⁸⁷⁰ Moreover, researchers who applied to the Nutrition Foundation for funding of their study had to submit their study design to the Foundation for approval. Although the Nutrition Foundation's stated intent was to 'assure the double-blind nature of their observations', many of the trials they did fund had other methodological problems, including using the Foundation's relatively low-dosage challenge cookie.⁸⁷¹ As demonstrated below, the manner in which many of these researchers interpreted their findings was also questionable, downplaying certain aspects of their study while emphasising others. Kaplan's interpretation of her trial's results, in contrast, was measured:

On the one hand, a much larger percentage of children responded to dietary intervention than found in previous studies. On the other hand, only half of the children who completed the study exhibited behavioral improvement, and it is safe to say that not a single parent believed that participation in this study had transformed their child into an easy to manage person. We removed everyday obstacles to compliance which practitioners regularly face: we determined the menus and provided the food at no cost to participants.⁸⁷²

Indeed Kaplan recalled that the 'parents' attitude was almost universally: "That's it? That's as good as it gets?" and: "Where can I get some Ritalin to try?" And it was so discouraging.⁸⁷³ Kaplan eventually left the area of research, partly because her colleagues dismissed her work 'as quirky', or unimpressive, but also because she found the lack of objectivity on both sides of the Feingold debate disheartening.⁸⁷⁴ Despite all the stated difficulties of designing a methodologically sound test of the Feingold diet, for Kaplan, creating a 'superb study' was an easier task than convincing physicians and parents that there was a link between food additives and behavioural problems.

⁸⁷⁰ For example, Cook, and Woodhill, 'The Feingold Dietary Treatment'; Arnold Brenner, 'A Study of the Efficacy of the Feingold Diet on Hyperkinetic Children: Some Favorable Personal Observations', *Clinical Pediatrics* 16 (1977), 652-6; R. C. Hindle and Janelle Priest, 'The Management of Hyperkinetic Children: A Trial of Dietary Therapy', *The New Zealand Medical Journal* 88 (1978), 43-5.

⁸⁷¹ NACHFA, *Final Report to the Nutrition Foundation*, 2.

⁸⁷² Kaplan, *et al*, 'Dietary Replacement', 17.

⁸⁷³ Bonnie Kaplan, Interview.

⁸⁷⁴ *Ibid*.

‘Arbitrary negative conclusions’? Interpreting the Trials of the Feingold Diet

In light of the methodological problems that hampered trials of Feingold’s hypothesis, one might expect that researchers, as well as those who reviewed their studies, would have been conservative with regards to interpreting their findings and making conclusive statements about the efficacy of the diet. Indeed, many researchers, recognising that their study, and most of the others, were not without methodological flaws, acknowledged that their results did not resolve the debate about the Feingold diet and, instead, suggested that more research be done to test the effects of food additives on behaviour.⁸⁷⁵ These calls for more research notwithstanding, researchers and reviewers differed drastically with regards to how they interpreted individual trials and the body of research as a whole. While some reviewers, such as psychiatrist Jeffrey Mattes, contended that ‘no single study has a reported a consistent dietary effect on the symptoms of the hyperkinetic syndrome’, others, such as Bernard Rimland, decried the ‘arbitrary negative conclusions’ reached by reviewers and researchers such as Mattes and argued that despite ‘anti-Feingold bias ... all studies, without exception, do concede that some children react to additives and some children do respond to the diet’.⁸⁷⁶ Such differences in interpretation suggest that those involved on both sides of the debate did not rely on science alone to make decisions about whether Feingold’s hypothesis was valid or not.

One of the best examples of how investigators could differ wildly in terms of interpreting their data can be found by comparing two well-cited trials, one led by psychologist J. Preston

⁸⁷⁵ For example, Williams and Cram, ‘Diet in the Management of Hyperkinesis’, 246-7; Geoffrey Thorley, ‘Pilot Study to Assess Behavioural and Cognitive Effects of Artificial Food Colours in a Group of Retarded Children’, *Developmental Medicine and Child Neurology*, 26 (1984), 56-61, at p. 56; K. S. Rowe, ‘Synthetic Food Colourings and “Hyperactivity”: A Double-Blind Crossover Study’, *Australian Paediatric Journal* 24 (1988), 143-7, at p. 144; L. Eugene Arnold, ‘Alternative Treatments for Adults with Attention-Deficit Hyperactivity Disorder (ADHD)’, *Annals of the New York Academy of Sciences* 931 (2001), 310-41, at p. 314.

⁸⁷⁶ Both reviews were published in the same volume of the *Journal of Learning Disabilities*. Mattes, ‘The Feingold Diet’, 321; Rimland, ‘The Feingold Diet’, 332.

Harley and the other led by toxicologist Bernard Weiss.⁸⁷⁷ Harley's trial, funded by the Nutrition Foundation, compared the effects of the Feingold diet and a control diet containing typical amounts of food additives on rates of hyperactivity in forty-six boys, thirty-six of whom who were aged between six and twelve years old and ten of whom were pre-school-aged. With the Foundation's funds (estimated at \$120 per family per week), Harley was able to design a trial that kept participants blind to the diets being tested and minimised violations.⁸⁷⁸ Included in these measures were a number of 'pseudo-dietary manipulations' that were employed in order to prevent participants from guessing which diet was being introduced, and challenge and food additive-free foods were produced and packaged to appear identical.⁸⁷⁹

In summarising his results, Harley stated that 'the overall results do not provide convincing support for the efficacy of the experimental (Feingold) diet'.⁸⁸⁰ But, despite this assessment, the results of Harley's trial were far more ambiguous. The most obvious problem was that Harley based his overall assessment on only one of his sample populations, namely, the thirty-six school-age boys, and downplayed the results of the smaller group of pre-schoolers. While the results from the older group were interpreted to be negative (and this was debatable – see below), those of the younger group appeared to provide solid support for Feingold's hypothesis. As reported by Harley: 'All ten mothers and four of the seven fathers of the pre-school sample rated their children's behavior as improved on the experimental diet.'⁸⁸¹

⁸⁷⁷ Harley, *et al*, 'Hyperkinesis and Food Additives'; Weiss, *et al*, 'Behavioral Responses'.

⁸⁷⁸ The amount of funding was not listed in Harley's published reports, but in Werry, 'Food Additives and Hyperactivity', 282.

⁸⁷⁹ Harley, *et al*, 'Hyperkinesis and Food Additives' 821.

⁸⁸⁰ *Ibid*, 826.

⁸⁸¹ *Ibid.*, 825.

Harley admitted that his interpretation was problematic, stating that ‘the attentive reader of this report has undoubtedly sensed, if not specifically identified, our discomfort and uncertainty in the manner of presenting the results on the preschool sample’.⁸⁸² The reasons Harley provided for ignoring the results of the younger group, primarily that they were only based on parental, rather than parental *and* teacher, rating scales, and that it was more difficult to gauge hyperactivity in preschoolers, however, begged the question of why, in such a carefully designed study, were younger children included if they were so difficult to test? The discounting of the younger group becomes more troubling when one considers Feingold’s observation that younger children were particularly susceptible to food additives, an observation that other researchers echoed.⁸⁸³ Indeed, other observers, including Bernard Weiss and C. Keith Conners, had trouble with the Wisconsin group’s lack of emphasis on the preschool sample. For example, Conners, whose opinion about the Feingold diet during the late 1970s could be best described as ambivalent, stated that ‘they cannot have it both ways. If their study did indeed rigorously achieve a complete disguise of the dietary manipulations, then the parent ratings, regardless of their “subjectivity” have to be explained. The probability of obtaining such findings by chance alone is miniscule.’⁸⁸⁴ Such criticisms notwithstanding, most later reviewers nevertheless concluded that Harley’s trial yielded little evidence in favour of Feingold’s hypothesis.⁸⁸⁵

Although the issue of the preschool data was the most questionable aspect of Harley’s study, other details relating to Harley’s interpretations highlight differences in how researchers described trial results. Weiss, for instance, not only disputed how Harley dealt with his

⁸⁸² *Ibid.*, 826.

⁸⁸³ Feingold, ‘Hyperkinesis and Learning Disabilities’, 800; Williams and Cram, ‘Diet in the Management of Hyperkinesis’, 245-6; Thorley, ‘Pilot Study’, 56.

⁸⁸⁴ Conners, *Food Additives and Hyperactive Children*, 39.

⁸⁸⁵ Werry, ‘Food Additives and Hyperactivity’, 282; Williams and Cram, ‘Diet in the Management of Hyperkinesis’, 244; Mattes, ‘The Feingold Diet’, 319; Bernard Weiss, ‘Food Additives as a Source of Behavioral Disturbance in Children’, *Neurotoxicology* 7 (1986), 197-208, at p. 200.

preschool sample, but his interpretation of the data generated from the older group also differed considerably. In describing how he interpreted the overall results, Harley stated that the ‘few significant findings related to diet that did emerge must be conservatively interpreted for several reasons’.⁸⁸⁶ One of the reasons Harley listed for his reticence was that the most positive ratings emerged from parents, not teachers.⁸⁸⁷

In contrast, Weiss had little trouble with the parental ratings, stating that thirteen of the thirty-six mothers and fourteen of the thirty fathers ‘recorded substantially improved behavior on the experimental compared to the control diet’, and added that six of the thirty-six teachers also reported less hyperactive behaviour when the boys were on the experimental diet.⁸⁸⁸ Furthermore, Weiss questioned the low frequency of observations, contending that this emphasised the relevance of dietary infractions, which occurred 0.65 times per week in the older group, and stressed that, when the entire sample of forty-six boys was considered, it had to be recognised that half of the mothers indicated that their sons had improved on the Feingold diet.⁸⁸⁹ In explaining why he thought Harley viewed his results so negatively, Weiss went as far as to suggest that to interpret them otherwise would have embarrassed his funders, and that the entire situation was ‘a salient example of the extra-scientific barriers posed to the Feingold hypothesis’.⁸⁹⁰

In order to explain why Weiss interpreted Harley’s trial so differently, it is helpful to consider his own conclusions regarding a trial that he himself conducted in 1980. Weiss received a grant from the FDA to test 22 children between two and a half and seven years old for eleven

⁸⁸⁶ Harley, *et al*, ‘Hyperkinesia and Food Additives’, 826.

⁸⁸⁷ *Ibid*.

⁸⁸⁸ Bernard Weiss, ‘Food Additives and Environmental Chemicals as Sources of Childhood Behavior Disorders’, *Journal of the American Academy of Child Psychiatry* 21 (1982), 144-52, at p. 144-5.

⁸⁸⁹ *Ibid*, 145.

⁸⁹⁰ Weiss, ‘Food Additives’, 200. Weiss proceeded to re-interpret a number of other trials which were thought to have yielded negative results and deemed instead that these also lent support to Feingold’s hypothesis. Weiss, ‘Food Additives’, 145-51.

weeks, and published his results in *Science*.⁸⁹¹ On the surface, the toxicologist's results were even less impressive than those of Harley; only two of the group demonstrated reactions to the challenge. Moreover, the parents of the children in Weiss' study had all previously reported reductions in their children's hyperactivity after they started the Feingold diet, thus calling into question their observations of their children's behaviour, and possibly Feingold's observations as well.⁸⁹²

Despite his seemingly unimpressive results, however, Weiss believed that his results supported Feingold's hypothesis. His justification for stating so was partly based in his background in toxicology. Unlike other researchers, who questioned Feingold's claims that a high percentage of hyperactive children reacted negatively to food additives, Weiss was only interested in whether or not such reactions were possible at all in any children. His study, therefore, was not intended to be 'a group experiment, but 22 separate experiments. Our aim was not to estimate population prevalence or sensitivity, but simply to determine if behavioral sensitivity to color additives could be detected in a controlled trial.'⁸⁹³ The response of one of Weiss' participants, in particular, provided convincing evidence for him that food colours could indeed evoke troubling behaviour in children:

One child reacted dramatically. This 34-month-girl, weighing about 13 kg, ... behaved significantly worse after challenge than after placebo on five of the seven aversive behaviors and on all of the global measures. One intriguing aspect of this child's response was her mother's ability to discriminate the response to color. She volunteered the information ... that her daughter had received the challenge six times during the 77-day period. She was correct five times. ... These data further strengthen the accumulating evidence from controlled trials, supplemented by laboratory experiments that modest doses of synthetic colors, and perhaps other agents excluded by elimination diets, can provoke disturbed behavior in children.⁸⁹⁴

⁸⁹¹ Weiss, *et al*, 'Behavioral Responses'.

⁸⁹² On the other hand, none of the children had been officially diagnosed as hyperactive, and none had exhibited 'clinically significant medical or psychiatric problems'. The group, therefore, might not have been one that reacted particularly strongly to food additives. *Ibid.*, 1487.

⁸⁹³ *Ibid.*, 1488.

⁸⁹⁴ *Ibid.*

Given the girl's young age, and the fact that the other strong reactor was only three-years-old, Weiss believed that his results especially highlighted the possible effects of food additives on younger children, a conclusion shared to differing degrees by many other investigators and Feingold himself.⁸⁹⁵

Although Weiss believed that his trial yielded support for the Feingold diet, many of Feingold's critics counted his trial among the negative results.⁸⁹⁶ In contrast, reviewers who supported Feingold, including Weiss, believed that Harley's results were favourable.⁸⁹⁷ Indeed, Weiss reviewed many of the most highly regarded trials of Feingold's theory in 1982 and concluded that all of them provided some support, concluding that:

The Feingold hypothesis points to new and potentially fruitful research areas for the etiology of hyperactivity and other behavior disorders which, in turn, enhance our understanding of brain-behavior relationships. . . . Specialists in child behavior should be alert to environmental contaminants as one of the potential contributors to the genesis of disturbed behavior.⁸⁹⁸

Weiss' assessment differed greatly from that of Mattes, who saw little positive emanating from Feingold's hypothesis:

this review illustrates the need for controlled objective investigation of any treatment intervention, no matter how enthusiastically endorsed. This area may well be a good example of how long research can continue on the basis of a popular 'fad' and chance positive results. The popularity of the Feingold diet might be seen as an outgrowth of sociological factors (eg., the desire for 'naturalness,' and suspicion of an 'establishment' which includes large food manufacturers) rather than true beneficial results. Clearly there is no rationale for being an advocate *for* artificial food colorings; these additives serve no function except cosmetic. But concern regarding their effects on the behavior and learning of children seems to be unwarranted.⁸⁹⁹

⁸⁹⁵ Feingold, 'Hyperkinesis and Learning Disabilities', 800; Goyette, *et al*, 39-40; Williams and Cram, 'Diet in the Management of Hyperkinesis', 244-6; Weiss, 'Food Additives', 151.

⁸⁹⁶ NACHFA, *Final Report to the Nutrition Foundation*, 24; Mattes and Gittelman, 'Effects of Artificial Food Colorings', 715.

⁸⁹⁷ Rimland, 'The Feingold Diet', 331; Weiss, 'Food Additives', 144-5.

⁸⁹⁸ A similarly-worded assessment can be found in Gladys Witt Strain, 'Nutrition, Brain Function and Behavior', *Psychiatric Clinics of North America* 4 (1981), 253-68; Weiss, 'Food Additives', 151-2.

⁸⁹⁹ Mattes, 'The Feingold Diet', 322.

Published after most of the trials testing Feingold's hypothesis were completed, and around the time of Feingold's death, these differing assessments highlight how divided many scientists remained even after the theory was tested.

It is hard to determine exactly why reviewers arrived at such different conclusions.

According to Ellen Silbergeld, an environmental health researcher who participated with NACHFA and led research linking lead exposure to hyperactivity, at issue for the Advisory Committee was the idea that the Feingold diet was a 'cure' for hyperactivity. This notion, which was not necessarily what Feingold originally advocated, and is certainly not what FAUS claims today, nevertheless bothered many mental health professionals who worked on hyperactivity.⁹⁰⁰ Indeed the language used by certain researchers and reviewers suggest that Feingold's hypothesis, and his manner of disseminating it, was galling to many. New Zealand child psychiatrist, John Werry, in response to a study published in the *Medical Journal of Australia* by child psychiatrist Peter Cook and dietician Joan Woodhill, strongly expressed how:

the most chilling aspect of Feingold's work lies in the enthusiasm with which it has been embraced by the anti-medication, anti-psychiatry section of the American public and used as a cudgel to try to close down paediatric psychopharmacological research in that country. The irony is that, if research with children is shut out in America, present clinical misuse of psychotropic drugs will continue unabated and unevaluated. Furthermore, who will then know which prophet, whether it be Feingold or some other, to follow, and public and profession alike will be at the mercy of every passing medical Pied Piper.⁹⁰¹

A series of letters responding to both Cook and Woodhill's study and Werry's response proceeded to inundate the *Medical Journal of Australia*, and reflected the fervour of both Feingold's supporters and detractors. Representatives of the food industry in Australia also engaged in the debate, calling for 'a balanced view of the Feingold hypothesis' to be given to

⁹⁰⁰ Ellen Silbergeld, Telephone Interview, 3 February 2009.

⁹⁰¹ Werry, 'Food Additives', 282.

the Australian public.⁹⁰² According to the authors of a generally supportive clinical report, which Werry and another author also lambasted, Feingold's manner of promoting his diet was much to blame for the uproar:

Unfortunately Dr. Feingold must bear much of the responsibility for such reactions - as by his unusual advocacy of his dietary programme he has actively alienated those of his colleagues who are best placed to evaluate it, and Professor Werry's emotive charges of quackery and the implication that the regime may be dangerous must be seen in that light.⁹⁰³

Similarly caustic debates also erupted in the pages of other journals and observers noted how emotion had supplanted reason on both sides of the debate.⁹⁰⁴ While reviewers in *Pediatrics* warned that 'concerns about additives and hyperkinesis developed as a result of feelings, beliefs, fads, and emotions and had little to do with science', an anonymous editorial in the *Lancet* believed that passions had been aroused on both sides:

The dietary theory of hyperactivity has aroused strong emotions. Believers in the scientific method felt challenged by the speed of its public acceptance and the lack of objective evidence. The excellent results of trials in which children and parents knew the purpose of the dietary regimen had added to the enthusiasm of the proponents and the disquiet of the food industry.⁹⁰⁵

Finally, there was the possibility that the food, chemical and pharmaceutical industries influenced how researchers interpreted their findings, as well as the likelihood that profound

⁹⁰² Council of Australian Food Technology Association, Inc., 'Dr. Benjamin Feingold – Hyperactivity', *Food Technology in Australia* 29 (1977), 433.

⁹⁰³ Possibly stinging from Werry's rebuke of what were merely a series of clinical observations, the authors somewhat pointedly suggested that Professor Werry was best suited to pursue the issue of the Feingold diet further in controlled trials. This suggestion was somewhat ironic given the fact that Werry was loath to spend any time or resources testing Feingold's theory – see quotation in Chapter 5. Werry, 'Food Additives', 282; Hindle and Priest, 'The Management of Hyperkinetic Children: A Trial of Dietary Therapy', *The New Zealand Medical Journal* 88 (1978), 344-5; R. C. Hindle and Janelle Priest, 'Dietary Control of Hyperkinesis' *New Zealand Medical Journal* 88 (1978), 345; John S. Werry and M. G Aman, 'Dietary Control of Hyperkinesis', *New Zealand Medical Journal* 88 (1978), 297-8.

⁹⁰⁴ One particularly animated symposium on the Feingold diet in which Feingold himself participated was published in *Academic Therapy*. Sieben, 'Controversial Medical Treatments', 133-47; Robert Buckely, 'Hyperkinetic Aggravation of Learning Disturbance', *Academic Therapy* 13 (1977), 153-60; Allan Cott, 'A Reply', *Academic Therapy* 13 (1977), 161-71; Benjamin F. Feingold, 'A Critique of "Controversial Medical Treatments of Learning Disabilities"', *Academic Therapy* 13 (1977), 173-83.

⁹⁰⁵ C. Warren Bierman and Clifton T Furukawa, 'Food Additives and Hyperkinesis: Are There Nuts Among the Berries?', *Pediatrics* 61 (1978), 932-3; Anonymous, 'Feingold's Regimen', 617.

distrust of these industries on the part of certain researchers swayed their interpretations.⁹⁰⁶ Again, Feingold's approach was seen to have affected how industry responded. His book, for instance, was described by Weiss as 'a polemic, presenting a committed position, not a tentative scientific argument, and based on one physician's experience. It hardly endeared him to the food industry, which swiftly counterattacked.'⁹⁰⁷ Weiss proceeded to blame lack of interest in Feingold's theory following Feingold's death on 'an effective publicity campaign by the Nutrition Foundation, and because of their unfamiliarity with the pertinent literature'.⁹⁰⁸

Researchers on the other side of the debate also admitted that Feingold's hypothesis posed a threat to industry. Psychiatrists Morris Lipton, one of the two co-chairs of NACHFA, and James P. Mayo, for example, stated that Feingold's claims had 'major implications for the public health of children and for the food industry ... at worst, companies would be required to reveal their trade secrets'.⁹⁰⁹ Lipton and Mayo also agreed with Feingold's argument that food additive manufacturers were loath to reveal even the chemicals they currently used, let alone restrict their use.⁹¹⁰ Other observers suggested that the food industry could do a great deal to defuse the situation by voluntarily removing some of the more cosmetic additives, particularly the colours, from the food supply.⁹¹¹ This suggestion, however, was not taken up, and the emotive nature of the debate continued until Feingold's death.

⁹⁰⁶ For an analysis of how the contrasting opinions about sick building syndrome were also shaped by ideological and political factors, see: Michelle Murphy, *Sick Building Syndrome and the Problem of Uncertainty: Environmental Politics, Technoscience, and Women Workers* (Durham, NC: Duke University Press, 2006).

⁹⁰⁷ Weiss, 'Food Additives', 198.

⁹⁰⁸ *Ibid.*, 204.

⁹⁰⁹ Morris A. Lipton and James P. Mayo, 'Diet and Hyperkinesis – An Update', *Journal of the American Dietetic Association* 83 (1983), 132-4, at p. 132.

⁹¹⁰ Feingold, *Why Your Child is Hyperactive*, 89-90, 126; Lipton and Mayo, 'Diet and Hyperkinesis', 132.

⁹¹¹ Bierman and Furukawa, 'Food Additives and Hyperkinesis', 933.

Although the contentious nature of the Feingold diet helps to explain differences in how the trials to test the hypothesis were interpreted, other, more subtle, reasons might have also played a role in polarising such assessments. One factor was related to how scientists representing different disciplines conceptualised the potential risk from food additives.⁹¹² For Weiss, a toxicologist who often dealt with trace amounts of hazardous material and long-term pathological effects, such as cancer, the positive response of ten per cent of his sample was cause for concern. Clinical psychologists and psychiatrists who believed that hyperactivity was already treatable with stimulant drugs, and who might be more inclined to concentrate on associated social, educational or familial issues as supplementary factors, in contrast, were not as concerned with the suggestion that a small proportion of the overall hyperactive population were affected by food additives. Weiss also believed that he, as a toxicologist, was more concerned about preventative health and public health policy than most clinicians, describing how ‘it’s very hard to get practicing physicians to think about wide issues in public health and especially prevention. You know, there’s no money in prevention, who’s going to pay you? So that’s a very big problem.’⁹¹³

Feingold himself admitted in his last publication that ‘controversy revolves around numbers. The critics of the hypothesis contend that only a small number, perhaps 5-10% of children, react adversely to food additives and salicylates rather than the 50% favourable responses reported by me.’⁹¹⁴ But although such discrepancies might have mattered a great deal to clinicians, particularly those who disliked Feingold’s populist approach, quantifying the risk of food additives was less important if you were a parent of a hyperactive child. Conners,

⁹¹² Disciplinary differences also shaped how scientists understood sick building syndrome. While industrial hygiene experts rooted their understanding of chemical exposure in terms of levels of toxicity determined by laboratory investigations, popular epidemiologists, who could be laypeople, activists or sympathetic scientists, gathered information about chemical exposure by mapping the distribution of health problems in relation to the location of suspected pollutants. Murphy, *Sick Building Syndrome*, 81-110.

⁹¹³ Bernard Weiss, Interview.

⁹¹⁴ Feingold, ‘Role of Diet’, 164.

considering Weiss' extremely reactive subject in particular, contended that scientists 'might discount the significance for the population at large, but if the child were *my* 3-year-old, it wouldn't matter. I would still choose to eliminate the artificial colors.'⁹¹⁵ Determining how the results of a trial should be interpreted, therefore, depended considerably on how one defined risk, and this could be influenced by both professional remits and personal situations.⁹¹⁶

Although the trials undertaken to test the Feingold diet suffered from methodological problems and were interpreted in vastly different ways, most physicians and allied health professionals have assumed that they provided little evidence to support Feingold's hypothesis. Indeed, by 1980 NACHFA had already concluded that any positive response to the Feingold diet was evidence of placebo and that Feingold's claims had been clearly refuted.⁹¹⁷ Paediatrician Esther H. Wender, one of the chairs of NACHFA, added in a review that the apparent success of the diet highlighted 'the power of food to function as a conditioned stimulus' and provided suggestions to clinicians on how to ease parents away from the idea that food additives triggered hyperactivity.⁹¹⁸ Other industry-supported groups, such as the American Council on Science and Health, also concluded that the diet did not help hyperactive children.⁹¹⁹ Although the conclusions of the NIH Consensus Development Conference were more ambiguous, following Feingold's death, most 'investigators seemed to

⁹¹⁵ Italics in original. Conners, *Feeding the Brain*, 175.

⁹¹⁶ Most of the historical work on the risk of disease and public health policy has not considered how patients themselves have interpreted such risks, although work on patient activism has provided some insight into this. Michelle Murphy's work on sick building syndrome, for example, shows how patients were concerned about the risks posed by their working environment even when toxicologists were unable to detect what was making workers unwell. Murphy, *Sick Building Syndrome*, 57-110. For more on how changing notions of risk have affected public health policy, see: Ulrich Beck, *Risk Society: Towards a New Modernity* (London: Sage Publications, 1992); Rosenberg, 'Pathologies of Progress'; Rothstein, *Public Health and the Risk Factor*; Luc Berlivet, "'Association or Causation?'" The Debate on the Scientific Status of Risk Factor Epidemiology, 1947 – c. 1965', in Virginia Berridge (ed.), *Making Health Policy: Networks in Research and Policy after 1945* (Amsterdam: Rodopi, 2005), 39-74.

⁹¹⁷ NACHFA, *Final Report to the Nutrition Foundation*, 31-4.

⁹¹⁸ Wender, 'Food Additive-Free Diet', 42.

⁹¹⁹ American Council on Science and Health, *Food Additives and Hyperactivity* (Summit, NJ: American Council on Science and Health, 1984).

have lost interest' in testing the hypothesis.⁹²⁰ Special education specialists Kenneth A.

Kavale and Mark P. Mostert, for example, have recently asserted that:

the empirical evidence appears quite steadfast and suggests that artificial additives serve merely a cosmetic function with no negative effects on behavior or learning ... the use of the Feingold K-P diet was not predicated on research evidence, which was decidedly negative, but rather on ideological factors like the desire for a nonintrusive, natural intervention.⁹²¹

The details of the trials, particularly their conclusion and discussion sections, however, reveal that Feingold's detractors did not rely on empirical evidence alone to make decisions about his hypothesis. Rather, many of the most cited criticisms of the Feingold diet had more to do with other factors, including concern about how children would cope with being on a special diet, the level of nutrients in a salicylate-free diet and, most importantly, whether or not families could actually carry out such 'a difficult and exacting regimen'.⁹²² As Chapter 10 illustrates, many of these concerns contradicted the experiences of families on the Feingold diet.

⁹²⁰ Lipton and Mayo, 'Diet and Hyperkinesis', 133.

⁹²¹ Kenneth A. Kavale and Mark P. Mostert, *The Positive Side of Special Education: Minimizing its Fads, Fancies, and Follies* (Oxford: Scarecrow Education, 2004), 210. The authors relied largely on Kavale's own 'meta-analysis' of the trials of the Feingold diet. While such meta-analyses may indeed provide an indication of the direction of results in a series of trials, in the case of Feingold diet, it appeared to have been a fairly blunt instrument. This is not only because of the relatively small number of trials included in the meta-analysis - Kavale and his co-author, Steven R. Forness, considered twenty-three studies, and referred to them as a 'small number of studies' - but also because the plethora of methodological and interpretative problems were not taken into consideration. Kavale and Forness, 'Hyperactivity and Diet Treatment', 325.

⁹²² Feingold's critics, for instance, often charged that his diet was unsafe because it would leave children malnourished. While Feingold did advocate removing several fruits and a few vegetables out of the child's diet in the initial stages of his regimen, he strongly encouraged re-introducing these foods after a couple of months, since he believed children most often reacted only to food additives. As early as 1976, moreover, researchers were reporting that, although the Feingold diet was lower in nutrients, particularly Vitamin C, it still exceeded the Recommended Daily Allowances (RDA's) set out by the FDA. Moreover, a study in 1980 concluded that 'a diet free of artificial colors and artificial flavors does not significantly change the nutrient intakes of children'. Nevertheless, many commentators continued to warn about the nutritional deficiencies of the Feingold diet. Feingold, *Why Your Child is Hyperactive*, 170; Conners, *et al.*, 'Food Additives and Hyperkinesis', 164; Joanna Dwyer; Patricia H. Harper, Charles H. Goyette and C. Keith Conners, 'Nutrient Intakes of Children on the Hyperkinesis Diet' *Journal of the American Dietetic Association* 73 (1980), 515-20; David, 'Reactions to Dietary Tartrazine', 122; Wender, 'Food Additives', 1206; C. M. Carter, M. Urbanowicz, R. Hemsley, L. Mantilla, S. Strobel, P.J. Graham, and E. Taylor 'Effects of a Few Food Diet in Attention Deficit Disorder', *Archives of Disease in Childhood* 69 (1993), 564-8, at p. 568; Lynn Murphy, Email Interview, 24 July 2008.

Conclusion

Reading the medical literature associated with the Feingold diet, it is tempting to classify all researchers and reviewers as either supportive or critical. Indeed, most of those involved in the debates chose one side or the other and were reluctant to change their opinion.

Nevertheless, some researchers did change their position regarding the Feingold diet. The views of psychologist C. Keith Conners, for instance, fluctuated when Feingold's theory was being tested intensively, and have continued to do so. Conners' interest in Feingold's hypothesis stemmed naturally from his decades of groundbreaking experience researching the diagnosis and treatment of hyperactivity, and he was among the first scientists to receive funding to test it.⁹²³

At first, Conners' results, published in *Pediatrics*, were in support of Feingold's hypothesis, although the psychologist cautioned that, due to 'several features inherent in the present study which need further evaluation, more study was required before any firm conclusions could be reached'.⁹²⁴ To at least one reader, however, this was a slight underestimate of the study's limitations. James S. Miller, a physician from California, wrote to *Pediatrics* shortly after and charged that while the article was 'not the worst you have published, it is surely in active competition'.⁹²⁵ Miller's chief complaint was that Conners' trial was not controlled, adding sarcastically that following 'the same line of intellectual rigor ... I will bet the editorial board even money that if they send me the raw data for this article, I can establish a statistically valid correlation between a major conjunction of the planets and/or Keltic divinations using the tarot'.⁹²⁶

⁹²³ Conners received funding from both the National Institute of Education and NIMH. Conners, *Food Additives and Hyperactive Children*, xi.

⁹²⁴ Conners, *et al*, 'Food Additives and Hyperkinesis', 161.

⁹²⁵ James S. Miller, 'The Diet Wasn't Controlled', *Pediatrics* 61 (1978), 326-7, at p. 326.

⁹²⁶ *Ibid.*, 327.

Although no other observers were so harsh, and although he defended his work against Miller's accusations, Conners designed an additional trial to test Feingold's theory. The results of this trial were more ambivalent but, while Conners was again concerned about methodological problems, he nonetheless asserted that 'data firmly establish that artificial colors may be partially disruptive to younger children.'⁹²⁷ A few years later, however, Conners' opinion about Feingold's hypothesis, represented in a book he wrote on the subject, had changed. Stressing that new 'ideas in behavioral science are often difficult to track down and evaluate', Conners warned that 'the "dramatic" nature of the effects has been grossly overstated by Dr Feingold, except insofar as placebo effects are dramatic among people who are at their wit's end with difficult and unmanageable children'.⁹²⁸

Conners' opinion would alter yet again. He returned to the subject of the Feingold diet in 1989 with *Feeding the Brain* and commented on how divisive the episode had been. For him, the entire controversy exemplified 'the deep distrust between practitioners who believe in the power of diet and scientists who regard it as fraud but who then go on to display bias in their own handling of the issues'.⁹²⁹ Moreover, he admitted that:

I have to admit that I have changed my mind about the Feingold diet since the 1970s. I sympathize with pediatricians and mental health workers who find the zeal of some patients for dietary treatments to be an impediment to other good treatments. I do not want to add to their burden. But my judgement is that the evidence is strong enough, at least for preschoolers, and especially those with confirmed allergic symptoms, that one should eliminate a broad range of unnecessary and possibly harmful ingredients from these children's diets.⁹³⁰

⁹²⁷ Goyette, *et al*, 'Effects of Artificial Colors', 40. A *Newsweek* report, however, stated that the results of this study were negative. Matt Clark, Dan Shapiro, Mary Hager, Janet Huck and Pamela Abramson, 'The Curse of Hyperactivity', *Newsweek*, 23 June 1980, 59.

⁹²⁸ Despite this conclusion, Conners graciously added that Feingold was owed 'a debt of gratitude for focusing attention on the research needed to advance in this area and to protect the heirs to our planet. The evidence has not been favourable to his hypothesis in our opinion, but his general advocacy on behalf of children deserves to be supported by all citizens through their support of efforts to increase research knowledge in this important area.' Conners, *Food Additives and Hyperactive Children*, 7, 107, 111.

⁹²⁹ Conners, *Feeding the Brain*, 3.

⁹³⁰ *Ibid.*, 184

Twenty years after this admission, however, Conners' position had changed for a final time. Now retired, and not completely familiar with the more recent trials of Feingold's theory, Conners stated in an interview that 'it didn't take too long before it became imminently clear that most of the results, with the possible exception of preschoolers, were not due to additives, but were placebo effect.'⁹³¹ As a caveat, however, Conners added that, when he saw patients, he nonetheless advised mothers of young children to avoid certain additives as 'a general kind of precaution because it was never firmly established that these preschool effects weren't there'.⁹³²

Unlike Conners, most researchers had little trouble coming to firm conclusions about the Feingold diet based on the trials and, subsequently, praising Feingold or condemning him. Considering the litany of methodological and interpretative problems that plagued the trials, how can this be explained? On the one hand, it is apparent that researchers were influenced by a large number of ideological, epistemological, economic and political issues, issues that have often shaped how scientists have made decisions about contentious theories.⁹³³ On the other hand, the history of how Feingold's theory was tested also reveals some of the limitations of using double-blind controlled trials as a tool to prove definitively the validity of some medical claims. It suggests that over-reliance on such trials for epistemological proof leads physicians away from other potentially fruitful sources of evidence, sources that at least could be used in conjunction with the findings of double-blind trials in the resolution of debates. As the next chapter attempts to demonstrate, the most important of these supplementary sources of evidence was to be found in the homes of Feingold families themselves.

⁹³¹ C. Keith Conners, Telephone Interview, 14 January 2009.

⁹³² Ibid.

⁹³³ For a few other examples of how similar factors have affected the outcome of debates in medical history, see: Tauber, *The Immune Self*; Pressman, *Last Resort*; Söderqvist, *Science as Autobiography*; Murphy, *Sick Building Syndrome*; Bivins, *Alternative Medicine?*; Dyck, *Psychedelic Psychiatry*.

Chapter 10

Feingold Families

Regardless of the conclusions reached by medical researchers about Feingold's theory, the ultimate arbiters of whether the Feingold diet worked or not were hyperactive children and their parents. Parents had to decide to attempt the diet, and subsequently adjust their shopping, meal-planning and cooking, monitor their children for compliance and determine if the diet worked or not. Their children, meanwhile, had to agree to the new dietary regimen, refraining from the processed foods, particularly snacks, drinks and desserts, that they had previously enjoyed and resisting pressure from peers to surreptitiously eat such items. In some ways, therefore, the greatest barrier to acceptance of Feingold's hypothesis was not the reluctance of physicians to support his theory, but the ability of parents and children to employ it.

This chapter explores why families decided to try the Feingold diet, how families employed the diet, whether or not families found the diet to be successful and what the experiences of families on the Feingold diet suggest about the role of patients and their families in informing debate about medical controversies. The primary source material for this chapter is thirty oral history interviews, including twenty-three interviews of parents, nearly all of whom were mothers, and seven interviews of grown-up children, nearly all of whom were male. Most of the interviewees were Americans representing most regions of the country, although there were also two Canadian and two British individuals interviewed as well. The interviews included not only those who had first learned about the Feingold diet during the 1970s, but also included those who used it during the subsequent three decades, in order to gauge how

experiences of Feingold families changed during the thirty-five years since Feingold wrote *Why Your Child is Hyperactive*.⁹³⁴

Once derided by A. J. P. Taylor (1906-1990) as being nothing more than ‘old men drooling about their youth’, oral history has become a crucial methodological tool for historians who wish to uncover patient experiences of illness and medical treatment.⁹³⁵ Although the situation has improved for more recent history, the voices of patients and their families remain largely absent from the history of medicine and, particularly, the history of disability and mental illness.⁹³⁶ When patients are the focus of historical inquiry, patient records written by physicians are often the primary source of information, although diaries, descriptions and depictions of patient experience found in literature and newspaper and magazine stories may also illuminate patients’ experiences.⁹³⁷ While such sources must be sufficient for historians working on earlier periods, for those working on twentieth-century history, evidence from

⁹³⁴ More details about how the interviews were conducted can be found in Chapter 1.

⁹³⁵ A. J. P. Taylor quoted in Brian Harrison, ‘Oral History and Recent Political History’, *Oral History* 1 (1972), 30-46, at p. 46. For an account of how oral history has developed as a historical technique, see: Paul Thompson, *The Voice of the Past: Oral History*, 2nd Ed. (Oxford: Oxford University Press, [1978] 1988). An introduction to the use of oral history in medical history can be found in Paul Thompson and Rob Perks, *An Introduction to the Use of Oral History in the History of Medicine* (London: National Life Story Collection, 1993).

⁹³⁶ There are exceptions to this, including: Steve Humphries and Pamela Gordon, *Out of Sight: The Experience of Disability 1900-1950* (Plymouth: Northcote House, 1992); Jocelyn Goddard, *Mixed Feelings. Littlemore Hospital - An Oral History Project* (Oxford: Oxfordshire County Council, 1996); Rebecca Fido and Maggie Potts, ‘Using Oral Histories’, in Dorothy Atkinson, Mark Jackson and Jan Walmsley (eds.), *Forgotten Lives: Exploring the History of Learning Disability* (Kidderminster: British Institute of Learning Disabilities, 1997), 35-46; Gittins, *Madness in its Place*; Kerry Davies, ‘“Silent and Censured Travellers?” Patients’ Narratives and Patients’ Voices: Perspectives on the History of Mental Illness Since 1948’, *Social History of Medicine* 14 (2001), 267-92; Liz Linthicum, ‘Integrative Practice: Oral History, Dress and Disability Studies’, *Journal of Design History* 19 (2006), 309-18; Jan Walmsley, ‘Life History Interviews with People with Learning Disabilities’ in Rob Perks and Alistair Thomson (eds.), *The Oral History Reader*, 2nd Ed. (New York: Routledge, [1998] 2006), 184-97, at p. 185.

⁹³⁷ Understandably, accounts of mental illness from those who were rich and famous are more common than those from the poorer classes. Dale Peterson, *A Mad People’s History of Madness* (Pittsburgh: University of Pittsburgh Press, 1982); Roy Porter, *A Social History of Madness: Stories of the Insane* (London: Weidenfeld and Nicolson, [1987] 1989). For some examples of health and illness in literature, see: Iain Bamforth (ed), *The Body in the Library: A Literary History of Modern Medicine* (London: Verso, 2003). On the limitations of patient records as an archival source, see: Jonathan Andrews, ‘Case Notes, Case Histories, and the Patient’s Experience of Insanity at Gartnavel Royal Asylum, Glasgow, in the Nineteenth Century’, *Social History of Medicine* 11 (1998), 255-81. Finally, photographs can also provide information about patient experiences. Mark Jackson, ‘Images from the Past: Using Photographs’, in Dorothy Atkinson, Mark Jackson and Jan Walmsley (eds.), *Forgotten Lives: Exploring the History of Learning Disability* (Kidderminster: British Institute of Learning Disabilities, 1997), 65-74.

interviews can provide insights that transform how the history of health and illness is understood. When oral history is ‘woven into broader historical analysis’, a more comprehensive picture of the history of health and illness is possible and, in some cases, the evidence provided can challenge prior assumptions about not only patient experiences, but also the development of medical knowledge.⁹³⁸

There are, however, challenges to conducting oral history interviews and interpreting such evidence. According to sociologist Trevor Lummis, the validity of oral history sources is determined by ‘the degree to which any individual interview yields reliable information on the historical experience, and the degree to which that individual experience is typical of its time and place’.⁹³⁹ Such challenges have been addressed by historian Kate Fisher, for example, in her work on the history of sexuality in Britain. In her case, the sensitive and, possibly, embarrassing nature of her interviews not only raised the issue of whether or not her subjects fully disclosed or remembered details about their sexual experiences accurately, but also highlighted concerns about how representative the interviewees were of the general population.⁹⁴⁰ Fisher contends, however, that ‘the perceived limitations of oral history – small sample size, lack of representativeness, the erosion of memory, and the impact of external influences on the construction of material – should not be viewed so negatively’ and can in fact be ‘key indicators of the meaning of experience’.⁹⁴¹ The methodology employed in the oral histories of the Feingold families has been similarly intended to balance the desire for accurate descriptions of events with the need to analyse why people responded in the way

⁹³⁸ Roy Porter, ‘The Patient’s View: Doing Medical History from Below’, *Theory and Society* 14 (1985), 175-98, at pp. 175-6; 193-4; Davies, ‘Silent and Censored Travellers?’, 291; Kate Fisher, *Birth Control, Sex and Marriage in Britain 1918-1960* (Oxford: Oxford University Press, 2006), 4.

⁹³⁹ Trevor Lummis, ‘Structure and Validity in Oral Evidence’, in Rob Perks and Alistair Thomson (eds.), *The Oral History Reader* (London: Routledge, 1998), 273-83, at p. 273.

⁹⁴⁰ Fisher, *Birth Control, Sex and Marriage*, 13.

⁹⁴¹ *Ibid.*, 13-4.

they did, and what this implies about how patients and their parents conceptualised their experiences.

In spite of these efforts, issues related to validity and interpretation of oral history evidence emerged in the interviews of Feingold families. The accuracy of anecdotes and memories of interviewees, for example, was a matter that required careful consideration, particularly with respect to interviews of adults who had been on the Feingold diet as children. While some interviewees could remember a great deal about their childhood experiences before and after the introduction of the diet, others struggled to do so. Nevertheless, each interview yielded some information about how children experienced the Feingold diet and, when viewed alongside parental testimony, interesting patterns surfaced.

In the case of parents, other issues emerged, particularly with respect to how parents might transform rather banal family memories into more profound family myths or traditions. Historian Ruth Finnegan has described how ‘those who enunciate and guard the traditions are not just passive transmitters but, also in a way, active creators of a family’s ethos’.⁹⁴² Although it can be difficult to distinguish an accurate retelling of an incident from an exaggeration or colouring of the event, measures were taken to guard against such occurrences or, instead, use them in a productive fashion.

For instance, interviews of mothers who had volunteered for FAUS were interpreted slightly differently than those of parents who had no formal connection to the association. Mothers

⁹⁴² Ruth Finnegan, ‘Family Myths, Memories and Interviewing’, in Rob Perks and Alistair Thomson (eds.), *The Oral History Reader*, 2nd Edition (New York: Routledge, [1998] 2006), 177-83, at p. 178. Historian Flurin Condrau has also discussed the need to recognise ‘the charm of the sources’ in patient-centred history. Condrau acknowledges that patient-generated accounts are compelling and add to our understanding of the history of illness and health, but urges historians to analyse contextually what it means to be a patient and to use patient accounts to inform broader debates about the history of medicine. Flurin Condrau, ‘The Patient’s View Meets the Clinical Gaze’, *Social History of Medicine* 20 (2007), 525-40, at p. 536.

who were active with FAUS, specifically Jane Hersey, Lynn Murphy and Shula Edelkind, not only had more reason to make their story sound dramatic or impressive, they also had repeated their story numerous times; indeed, Hersey had written a book that described many of her experiences with the Feingold diet.⁹⁴³ It is possible that their recollections were moulded over the years to fit an idealised depiction of life on the Feingold diet. Although the fact that these accounts might have been embellished resulted in a more cautious approach to interpretation, it also proved to be useful in other respects. It was helpful, for example, to have a somewhat idealised version of a family's experience on the Feingold diet to compare with other families' experiences. Anecdotes that appeared overly-rehearsed or somehow mythologised were also interesting since they represented aspects of the diet that a mother wanted to emphasise, often because her experiences contrasted with what was reflected in the media or in the medical literature. In other cases, the testimony of the mother could be compared against her child's account of events.

Another issue regarding the oral history evidence concerned how representative the sample was of families who had tried the Feingold diet. It is clear that a sample of thirty people cannot adequately represent the overall experiences of the tens of thousands of families who tried the Feingold diet. Furthermore, it could be argued that the group interviewed were somewhat self-selected, representing families who were more successful with the diet. These criticisms, however, are only partly accurate. First, the sample was recruited in a variety of ways, including advertisements in *Pure Facts*, FAUS's newsletter, referrals from participants and even chance encounters with those who had experienced the diet; some interviewees,

⁹⁴³ Jane Hersey, *Why Can't My Child Behave?* (Williamsburg, Virginia: Pear Tree Press, Inc., [1996] 2006). Similarly, accounts of the Feingold diet that were published on the FAUS website or in their newsletter, *Pure Facts*, were interpreted differently than the oral accounts. Whereas the historian can ask supplemental questions of an interviewee to clarify or confirm, this is obviously not possible with written accounts, such as those found in *Pure Facts*. Although *Pure Facts*, which usually includes a success story each issue, was examined, the examples provided in this chapter are from oral interviews. In a few cases it was possible to compare interviews with a success story in *Pure Facts* and, although there were no glaring inconsistencies, it was clear that the interviews provided considerably more information.

therefore, were not self-selected. Moreover, a number of participants volunteered even though their children ultimately decided not to follow the Feingold diet. Finally, despite the relatively small sample size, certain patterns and characteristics emerged within a population that was, in other respects, quite heterogeneous. Although a larger number of interviews might have reinforced the existence of such patterns, it is questionable whether it would have revealed important trends that were not detected in this sample.⁹⁴⁴

The experience of families that employed the Feingold diet is not only an essential aspect of the history of Feingold's theory, but it also demonstrates how patient experience affected the development of medical knowledge during the late twentieth century. In particular, the experiences of Feingold families demonstrate that physicians who warned that the Feingold diet was virtually impossible to employ were incorrect. Although families found that the diet was difficult, and many were not able to persevere with it, others did succeed, often despite the lack of support from medical professionals. Typically, though not exclusively, the more successful families were those in which the parents were married, educated, financially secure, and in which the mothers, in particular, demonstrated the diligence, assertiveness and observational skills necessary to stick to the diet, ensure that school authorities, relatives and friends of the family adhered to their dietary wishes and determine for themselves whether or not the diet was effective. Moreover, the success of many Feingold families, often over a period of decades, suggests that the results of double-blind trials should not have been the only way to assess the efficacy of the Feingold diet, and that improved understanding of patient experiences can inform the development of medical knowledge and health policy.

⁹⁴⁴ This is not to say, however, that another historian would not interpret the interviews differently or identify other patterns or trends, depending on their background, experiences and personal beliefs about hyperactivity, food and medicine.

This chapter opens by exploring how and why parents made the decision to try the Feingold diet. It reveals that families came to Feingold's hypothesis from a variety of viewpoints; while the theory fitted in with the preconceived notions of some parents, it represented a radical idea to others. The difficulties Feingold families faced attempting to employ the diet are then examined. Parents, as well as their children, faced different challenges in adhering to the diet, but what their accounts indicate is that the Feingold diet was by no means an impossible imposition, as many of Feingold's critics charged. The chapter concludes by discussing how the experiences of parents who found success with the Feingold diet demonstrate that medical expertise was not limited to physicians and other health professionals. This being the case, the observations and opinions of Feingold families should have had a larger role in resolving the debates about the Feingold diet.

**‘Sounds like a lot of new-age hooey, but I’ll try anything to help the boy’:
Deciding to Try the Feingold Diet**

Many of the reasons why parents decided to try the Feingold diet have been discussed briefly in Chapters 6 and 7. Frustration with treatment alternatives for hyperactivity and concern about the food supply spurred numerous parents to seek out other solutions. But it is also important to note that parents who turned to the Feingold diet did so for a variety of specific reasons and in the midst of differing circumstances. While some parents found out about the Feingold diet soon after their child's behaviour had become problematic, others had endured years of attempting various treatments unsuccessfully. Equally, the notion that food additives could affect behaviour fitted neatly into the ecological ethos of some parents, but seemed preposterous to others. Parents also learned about the diet from a wide range of sources, sometimes when they were actively looking for an alternative, and other times in a more serendipitous fashion.⁹⁴⁵ These differences in how parents discovered the Feingold diet and

⁹⁴⁵ Although many families found out about the Feingold diet through word of mouth and chance encounters with people already on the diet, others learned about it via a variety of media sources. Not surprisingly, none of those interviewed found about the diet through medical journals. Families could find out about some

decided to try it not only highlight how, in some respects, Feingold families were a diverse group, but also how unconventional medical ideas reached patients via many different routes. It would be a mistake, therefore, to assume as some do that Feingold families were all living an organic, bohemian lifestyle and that they all patronised alternative health practitioners. Although the families may now share certain beliefs and values about nutrition and psychiatry, having employed the Feingold diet for many years, it is clear that they did not all do so when they first heard of it.

Parents differed, for instance, with regards to their experience of using other treatments to treat their children's behavioural problems. For a number of parents, such as Michigan mother Lora Hollins, stimulant drugs were simply not an acceptable treatment. Hollins recalled fighting with her son's school over whether or not her son should be prescribed Ritalin:

Back in the early 80s when I declined the absolute orders of the school that my child be put on Ritalin, they literally threatened that he'd have to be removed from school. ... And I just pushed right back because they just assumed that they were going to bully me like the rest of the little country girls in the small town I lived in here in Michigan. But I was not the quiet, gentle little country girl, I was a girl from over by Chicago. I just pushed them right back and said, 'If you decline to have him in school then perhaps the state will reimburse me to educate him.' And they just shut right up.⁹⁴⁶

Other parents, such as Alberta mother, Bonnie Kowaliuk, who was 'quite uncomfortable' with the possible side effects of stimulant drugs, also experienced pressure from her son's school, which wanted him to take Ritalin. Kowaliuk resisted their demands, stating that she and her husband would 'try everything we can versus doing, doing the amphetamines and the Ritalin to address his health needs'.⁹⁴⁷

aspects of the medical debates, however, through FAUS's newsletter, *Pure Facts*, which published summaries of research conducted to test Feingold's hypothesis.

⁹⁴⁶ Lora Hollins, Telephone Interview, 17 February, 2008.

⁹⁴⁷ Bonnie Kowaliuk, Telephone Interview, 5 November 2007.

In a number of cases where the children were not of school-age, parents who were hesitant about the use of amphetamines did not feel pressured by school authorities and had more time to explore their options. Although Californian Lynn Murphy described how she had prepared herself ‘for the possibility that he’d have to be on meds’ when her son went to school, she was grateful that she discovered the diet before he was prescribed anything, and the school never discovered that he had been diagnosed with hyperactivity.⁹⁴⁸ Michigan’s Lisa Manciewicz, on the other hand, was cautious about mainstream approaches to hyperactivity because of an experience involving her sister: ‘I had a sister that the teachers would chronically tell my mom that needed to be on medication, and they bothered her so much about it that she lied and said, “I put her on it.” And as soon as she said it, they said, “Oh, she’s doing great!”’⁹⁴⁹

In other situations, parents had tried other treatments, experienced limited success, and sought alternatives. Texan Marilee Rigg, for example, had noticed that Ritalin appeared to help the boy who lived next door, but found that the stimulant did not improve the behaviour of her son, Brian:

For this other child it was effective. And I think that for some children it is the answer, but for Bryan it was not the answer. He lost appetite, his behaviour was worse when he came off the cycle of Ritalin. I was very unhappy and I took him off.⁹⁵⁰

Other parents also found that hyperactivity drugs were either not helpful or had distressing side effects. A Virginia mother, Colleen Davis, described how she ‘tried Ritalin. It didn’t work. We tried Strattera. It didn’t work. And I ended up ultimately putting him on Concerta, but when he was on Concerta I noticed side effects – he had a real difficulty sleeping.’⁹⁵¹

Californian Susan Leitner did not want her son to be prescribed Ritalin, but acquiesced to the

⁹⁴⁸ Lynn Murphy, Email Interview, 24 July 2008.

⁹⁴⁹ Lisa Manciewicz, Telephone Interview, 17 April 2008.

⁹⁵⁰ Marilou Rigg, Telephone Interview, 20 May 2008.

⁹⁵¹ Colleen Davis, Telephone Interview, 5 February 2008.

wishes of his school to have him prescribed the drug. When her son was on the drug, however, neither she nor the officials at her son's school were satisfied with its effects, and she looked for alternatives, while the school officials tried to convince her to increase her son's dosage. Ironically, she was convinced to implement the Feingold diet fully – she had been casually experimenting with it – when visiting a shop in the basement of her son's therapist:

There was a little snack shop there and the kids were looking at the different snacks going, 'Can we have this? Can we have this? Is this for us?', and I was saying, 'No that has colour. No that has colour. Yeah that would be okay.' And this woman came over and said, 'Excuse me, are you using the Feingold program?' And that was my connection with somebody who had actually used it and who could talk to me about it.⁹⁵²

Reflecting on why she chose to try the Feingold diet, Marilee Rigg declared, 'when you're so desperate for help, you'll try anything'.⁹⁵³ Indeed, most parents expressed feelings of desperation and hopelessness prior to attempting the Feingold diet. For Taunya Stevenson, whose ten-year-old son's behaviour had regressed when he was prescribed Ritalin, and had also been receiving 'continual counselling', the Feingold diet was 'do or die'.⁹⁵⁴ Other parents, such as Shula Edelkind, who found out about the Feingold diet while living in New York, described how her son became:

more and more difficult to handle. He would scream at nothing. You could ask him if he wanted a scrambled egg or a fried egg, he would scream. He couldn't handle any kind of input. He couldn't handle any kind of choices. He would have tantrums from one end of the day to the next. He would roll on the floor and would scream. He'd climbed on the coffee table and I took him off and said, 'No, no!' And he did that 35 times in a row. And I sat him down so hard and I thought, 'Oh my God if I were stronger I would break his back', and I called Jewish Family Services and said 'You've got to help me before I kill this child.'⁹⁵⁵

⁹⁵² The woman who came up to Leitner was Lynn Murphy, another one of the mothers interviewed. Susan Leitner, Telephone Interview, 8 April 2008.

⁹⁵³ According to Marilee Rigg and her son, Brian, the greatest fear was that he would never finish school: 'My mom had nightmares of her twenty year old son being able to drive to his own 6th grade graduation.' Brian Rigg eventually earned a PhD in history from the Cambridge University. Brian Rigg; Marilee Rigg.

⁹⁵⁴ Taunya Stevenson, Telephone Interview, 29 March 2008.

⁹⁵⁵ Shula Edelkind, Interview.

Such desperation forced some families to embrace notions about nutrition and behaviour that they would have rejected before. When Sean Corr's wife Carrie put forward the idea of trying the Feingold diet, for example, he recalled how 'I was thinking to myself that this sort of sounds like a lot of new age hooley, but I'll try anything to help the boy.'⁹⁵⁶ As Carrie described, 'I was excited, Sean was the sceptic. We needed to do something to help Joshua and I was willing to try anything before medication.'⁹⁵⁷ Much like Sean Corr, Lynn Murphy was sceptical about Feingold's theory, and was hesitant to try his diet:

The first two times I heard it, I thought it could not possibly be the case because some of the symptoms were severe. I never gave food chemicals a thought. I was raised on them. ... I did nothing with the information for a year. I really figured it was an off-shoot of the health food movement in the 'hippy' days. However, we had no other options, so when he was about three, we tried it.⁹⁵⁸

Other mothers, such as Shirley Fadden, from Massachusetts, 'did not in a million years think diet would change my son', and felt that medication would likely be necessary for him at some point, but, since her husband was against medication, agreed to try the Feingold diet.⁹⁵⁹ Similarly, an anonymous mother described how, although she had never considered that food additives could cause health problems, 'I was so desperate, I'd try anything. I would not have disregarded any solution that anyone had given me as long as it was reasonable and safe.'⁹⁶⁰

In contrast, other parents found that Feingold's theory made sense, but often for different reasons. Lora Hollins, who described herself as 'a poor hippy living in the woods' during the early 1980s, had a longstanding interest in unprocessed food, shopped at a co-operative store that sold health foods such as 'organic flours and cold-pressed oils', and 'made a lot of stuff

⁹⁵⁶ Sean Corr, In-Person Interview, 6 December 2007.

⁹⁵⁷ Carrie Corr, Email Interview, 20 January 2008.

⁹⁵⁸ Lynn Murphy, Interview.

⁹⁵⁹ Shirley Fadden, Email Interview, 19 February 2008.

⁹⁶⁰ Anonymous, Telephone Interview, 17 January 2008.

from scratch'.⁹⁶¹ The fact that Hollins suffered from numerous allergies and believed that her asthma was related to food colours also predisposed her towards the Feingold diet. For other parents, such as Kelly Anne Tooker, who had been raised on the Feingold diet herself, avoiding food additives was self-explanatory: 'Why chance it? To the best of our understanding our brain chemistry is affected by the foods that we eat. If we're eating food with chemicals, we're affecting our brain chemistry. Why do that to a child? You want to choose foods in their most organic, natural state.'⁹⁶² Similarly, Canadian Tim Gooding, whose parents had immigrated to Ontario from the UK, recalled how his parents' longstanding interest in organic, home-grown food minimised the disruption of the Feingold diet to their household.⁹⁶³

Although some parents, such as Marta Phillips from Washington State, were also already interested in natural foods before learning about the Feingold diet, there were others who had quite different reasons for questioning food additives.⁹⁶⁴ For example, Paula Kimball, a Canadian who taught allied health sciences at a Texas university, was shocked in hindsight that her training as a histologist did not alert her to the dangers of certain chemicals. When she discovered that a number of foods she was feeding her son contained tartrazine, she recalled how:

I used to use tartrazine in the lab under a fume hood. ... Well it was like, duh? That's what I felt like. When I was reading the chemicals in some of these foods and the colours and what their colour names were, more than just Yellow number five, I read that it was tartrazine, I started thinking, we use these, these are hazardous materials in the lab. These things have MSDS [Material Safety Data Sheets] forms which I know because I used to work in histology labs and we were putting this in our mouths, we were eating this. So that's when I realised, you know, there's something wrong here and I should have known better.⁹⁶⁵

⁹⁶¹ Lora Hollins, Interview.

⁹⁶² Kelly Anne Tooker, Telephone Interview, 28 January 2008.

⁹⁶³ Tim Gooding, In-Person Interview, 4 February 2009.

⁹⁶⁴ Marta Phillips, Telephone Interview, 13 February 2008.

⁹⁶⁵ Paula Kimball, Telephone Interview, 4 February 2008.

Kimball's husband was also appalled 'that we were intelligent people that allowed this to happen, that we didn't read what we were putting into our mouths'. Similarly, both Susan Leitner and her son Benjamin agreed that, once Benjamin had begun to study chemistry at university, he was much more convinced that food additives could affect behaviour and was more willing to remain on the diet.⁹⁶⁶

Just as the families approached Feingold's idea from a range of perspectives, they found out about the Feingold diet in a variety of ways. As with Susan Leitner's experience in the shop in her son's therapist's office, many parents found out about the Feingold diet unexpectedly. Marilee Rigg, for example, was unaware of the diet until her then-husband brought a work colleague and his wife home for a meal. She turned out to be the president of the Feingold Association of Houston.⁹⁶⁷ Quite often parents were told of the diet by a mother who had successfully used the diet herself. Parents also heard word of the diet through relatives, friends, neighbours, magazines, television programmes, and, increasingly during the last decade, the internet.⁹⁶⁸

That parents learned about the Feingold diet in a wide variety of ways illustrates Feingold's success in promoting his diet directly to the general public, after his attempts to gain the approval of his fellow physicians failed. Interestingly, only one parent interviewed found out about the diet through a health professional, specifically a chiropractor.⁹⁶⁹ Indeed, when most

⁹⁶⁶ Susan Leitner, Interview; Benjamin Leitner, Telephone Interview, 22 April 2008.

⁹⁶⁷ Marilee Rigg, Interview.

⁹⁶⁸ Although the internet has provided parents access to much more information about alternatives to conventional medical treatment, as Rima Apple suggests, the number of choices can be overwhelming. Despite this, research by the Pew Internet and American Life Project suggests that health advice seekers who employ the internet are quite savvy and are relatively skilled at rejecting websites that seem too commercial or unprofessional. Rima Apple, *Perfect Motherhood: Science and Childrearing in America* (New Brunswick, New Jersey: Rutgers University Press, 2006), 140-1; Pew Internet and American Life Project, 'Vital Decisions: How Internet Users Decide What Information to Trust When They or Their Loved Ones are Sick' www.pewinternet.org/~media/Files/Reports/2002/PIP_Vital_Decisions_May2002.pdf.pdf, accessed 14 April 2009.

⁹⁶⁹ Leah Hause, Telephone Interview, 12 February 2008. Indeed, certain schools of chiropractics advocate the Feingold diet, and other alternative treatments for hyperactivity, in conjunction with chiropractic treatment.

parents mentioned the Feingold diet to their general practitioner, paediatrician or psychiatrist, they were warned that it was not recognised as an effective treatment for hyperactivity.⁹⁷⁰

The willingness of parents to ignore the advice of their physicians is similar to Rima Apple's findings regarding how mothers during the last third of the twentieth century were increasingly willing to question the advice of an 'autocratic health-care practitioner'.⁹⁷¹ By the 1970s mothers could choose from any number of health 'experts', but then had to make difficult decisions about which 'expert' to trust.⁹⁷²

Although Lynn Murphy's first general practitioner was supportive and 'interested in nutrition to begin with', her family had to change health insurance plans and all of the subsequent physicians they spoke to were dismissive: 'They would all recite the same phrases: "Dangerous for their nutrition", "Lack of Vitamin C", "No evidence that it works"... "It works in a very few cases."' ⁹⁷³ Similarly, Bonnie Kowaliuk's original physician was supportive, but then retired. Kowaliuk doubted that their 'new medical doctor... would be open to a lot of that. I mean you have to find a pretty special medical doctor who is open to it.' ⁹⁷⁴ When the psychiatrist Marta Phillips' son was seeing suggested that he be prescribed Ritalin, she said, "I'm not going to use the medication, we're going to use the diet instead", and the psychiatrist said, "Oh well I've heard that they don't work." I said, "That's interesting but we're going to try anyway." We've not been back to that psychiatrist. ⁹⁷⁵

William H. Koch, *Chiropractic: The Superior Alternative* (Calgary: Bayeux Arts, 1995), 95; Maura Roan and Jessica Roan, 'ADHD and Chiropractic', *Aspire Magazine* (August/September 2007), www.aspiremag.net/articles/parenting/childrenshealth/adhdandchiropractic.html, accessed 23 February 2009; Bonnie Kowaliuk, Interview; Lora Hollins, Interview.

⁹⁷⁰ As Apple contends, while physicians may value the information provided by parents regarding some aspects of health care, for example, the side effects of drugs, they can also feel that their medical authority is threatened when parents suggest the employment of alternative treatments, such as the Feingold diet. Apple, *Perfect Motherhood*, 162-3.

⁹⁷¹ Apple discusses how two women's health movements in particular, the Boston Women's Health Book Collective and La Leche League, provide examples of how women attempted 'to alter the authoritarian medical system they faced'. Apple, *Perfect Motherhood*, 134-6

⁹⁷² *Ibid.*, 136.

⁹⁷³ Lynn Murphy, Interview.

⁹⁷⁴ Bonnie Kowaliuk, Interview.

⁹⁷⁵ Marta Phillips, Interview.

Finally, Susan Leitner's paediatrician was more concerned about the practicality of the diet, warning her:

that there's not going to be anything you can eat except chicken breast and pineapple. I knew that wasn't true. He pooh-poohed it. Eventually, he did suggest it to people as something off the wall they could try. For years and years he said it was a really stupid idea and I was quite wacko.⁹⁷⁶

Leitner's willingness to go against her physician's advice was typical of many parents who decided to try the Feingold diet. Although the physicians of a few parents, such as Marilee Rigg, did not discourage them from employing the diet after it appeared to be working, others, such as Taunya Stephenson, had stopped 'seeing any doctors because they weren't helping'.⁹⁷⁷ In other words, many parents, often out of frustration, gave up on their physician's advice and decided that they would take primary responsibility for the health of their children.⁹⁷⁸ In so doing, parents demonstrated a willingness to defy medical authority and to take responsibility for their children's health. As the following paragraphs demonstrate, parents' defiant attitude was often supplemented with a resolute determination that their children follow the diet at home, school, at birthday parties, and anywhere else their children could come in contact with food additives.

'No big deal' or 'Very difficult'?: Adhering to the Feingold Diet

One of the chief criticisms of the Feingold diet was that it was too difficult for the typical American family to undertake.⁹⁷⁹ Conversely, some of Feingold's advocates have argued that the diet was not actually that difficult, and that most families could cope with it. Jane Hersey, for example, stated that 'trying to deal with a difficult (or impossible) child is what puts the

⁹⁷⁶ Susan Leitner, Interview.

⁹⁷⁷ Taunya Stevenson, Interview; Brian Rigg, Interview; Marilee Rigg, Interview.

⁹⁷⁸ In one case a parent was able to convince her physician to consider the diet: 'The doctor said as long as we weren't extreme about it ... he would go along with it, but he started to see big differences so he was impressed and wanted to use the diet on other of his patients and read all the literature about it.' Anonymous, Telephone Interview, 5 February 2008. For the most part, however, Feingold families received little co-operation from their physicians, in terms of helping them employ the diet.

⁹⁷⁹ See Chapter 9.

strain on the family. Changing some of your grocery brands is no big deal.⁹⁸⁰ The experiences of most Feingold families, with the possible exception of Hersey, however, suggests that adhering to the Feingold diet was arduous in many aspects, and that families who found success with the diet often shared a number of important attributes.

Once parents decided to attempt the Feingold diet, they were faced with a number of daunting challenges. These included convincing their children to follow the diet, changing their shopping and cooking practices and monitoring what their children ate and their resulting behaviour. The success families had in overcoming these challenges depended on a number of factors, including the degree to which the Feingold diet differed from their previous diet, how compliant friends, family, the school system and, most importantly, their children were with regards to the diet, and how able families were to observe their child's eating and behaviour, advocate for them and adapt the diet for their specific needs. Given the challenges inherent in the diet, it is perhaps unsurprising that two-parent families, particularly those in which the earnings of the father allowed the mother to stay at home for extended periods, tended to experience more success. Still, a number of single mothers were also able to persevere with the diet, despite its vicissitudes, suggesting that, while the Feingold diet was 'a difficult and exacting regimen', it was by no means an impossible one.⁹⁸¹

The first task all parents had in employing the Feingold diet was convincing their children, and the rest of the family, to try it. Despite the assertion made by a team of researchers that 'strict Feingold diet appears to be distasteful to the typical American child', children's

⁹⁸⁰ Jane Hersey, Email Interview, 1 July 2006. To a certain extent, Hersey may have been expressing how she felt about the difficulty of following the diet in the twenty-first century, compared to what it was like to attempt the diet in the 1970s and 1980s. Certainly, as Chapter 11 contends, parents found it easier to follow the diet in the 2000s, with expanded lists of 'Feingold-friendly' food, more organic and additive-free products and more support from FAUS and other parents via the internet. Nevertheless, in another interview, Hersey maintained that at 'first I found having to do the extra food preparation, it seemed like a chore, but compared to dealing with a child who was out of control, and a husband who was sick, it wasn't that big a deal'. Jane Hersey, Telephone Interview.

⁹⁸¹ Carter, *et al*, 'Effects of a Few Food Diet', 568.

responses to the prospect of a diet that eliminated food colours and flavours fluctuated from dread to optimism.⁹⁸² Certainly some children were highly resistant to the idea of avoiding food colours and flavours. An anonymous interviewee, whose family was already eating a fairly additive-free diet, for example, declared, ‘I hated the diet. Tomato sauce. Ketchup. Pizza. I didn’t have so many specific losses, but I remember feeling very much confined, bitter, and angry about the diet.’⁹⁸³

Other children disliked having to go on the diet at first, but recognised that it might be of some benefit. Ben Leitner, for example, expressed how the diet was ‘kind of annoying, but if it was going to help then, you know, back then I think it was annoying because it set me apart from the other kids, but if it was going to help me then it was worth it.’ Ben’s mother Susan reiterated this stating how at ‘first he was like, “I don’t want to do it, I don’t want to do it.” But okay, we’re not going to try to do it if you’re not going to buy into it because it won’t work. So, he decided he was going to do it.’⁹⁸⁴ Other children, such as Joshua Stephenson, were hesitant, but ultimately pliable: ‘I didn’t want to give up all the good food that I was eating, like the dyed foods and all that. I didn’t really like it, but I thought okay, I’ll go with it.’⁹⁸⁵ Similarly, an anonymous interviewee stated how, when his mother began the Feingold diet, he ‘didn’t know she was changing it at first but when I found out I wasn’t happy at first, but I thought it would make me better.’⁹⁸⁶

Finally, there were children who were almost eager to try the new regimen. Brian Rigg, for example, did not mind starting the diet, in part, because it meant an alternative to Ritalin, his feelings for which he described as follows: ‘I hated it. I remember Mom giving me the pill

⁹⁸² Gross, *et al*, ‘The Effects of Diets Rich in and Free from Additives’, 55.

⁹⁸³ Anonymous, Email Interview, 20 May 2008. Tomatoes were one of the salicylate fruits and vegetables that were banned on the first stage of the diet. Unfortunately, this interviewee reacted to them and grapes.

⁹⁸⁴ Susan Leitner, Interview.

⁹⁸⁵ Joshua Stephenson, Telephone Interview, 1 April 2008.

⁹⁸⁶ Anonymous, Email Interview, 7 February 2008.

when we were in the car and I started acting up and I hated the taste of it. ... I lost my appetite and I'd be really up and then really down. I do remember not liking that pill'.⁹⁸⁷ It also helped that Rigg's mother had studied home economics in college and 'was a phenomenal cook. So she was cooking things from scratch anyway because she liked doing it. So our diet was pretty good.'⁹⁸⁸

Just as children's opinions about the Feingold diet differed at first, so too did their parents' first experiences of attempting the diet. The first stage of the diet was quite restrictive, since it not only eliminated artificial colours and flavours, but also fruits and vegetables, such as tomatoes, apples and grapes, that contained high levels of salicylates. As Lisa Manciewicz described, 'stage one was very difficult because you are so limited because you are eliminating the salicylates. That was tough because that's in so many things naturally. I had to take away so many of his favourite foods.'⁹⁸⁹ Bostonian Heather Meath also found that the first stage of the diet was a challenge:

It was very difficult [sigh]. You know, three months of cleaning cupboards and not eating anything and trying to find the food you need ... Originally ... the meal prep was enormous. You know, making fake tomato sauce. My kids were onto me. They figured it out. So that part was tough ... I think the hardest thing was snacks. My children had been accustomed to eating certain kinds of snacks, junk food if you will, and, you know, they still wanted a little bit of that. I felt guilty that they couldn't have that and so that was tough.⁹⁹⁰

⁹⁸⁷ Brian Rigg, Interview.

⁹⁸⁸ Ibid. Brian Rigg and his mother, Marilee, had different recollections of how long it took the diet to be effective. While Brian Rigg believed that the effects were immediate, and remembered school reports confirming this, his mother recalled it taking 'six to eight weeks, really working with the diet closely to see positive effects for Brian'. Marilee Rigg, Interview.

⁹⁸⁹ Lisa Manciewicz, Interview. Most parents also thought that the Feingold diet was more expensive than their previous diet, although some believed that not buying as much convenience foods or eating at restaurants helped to lower their food bill. Colleen Davis. In general, most parents would have agreed with Bonnie Kowaliuk's statement that, in the 'short term, yeah it is expensive, but the long term benefits far outweigh the costs that you have to put out to ensure that your children are healthy and you are healthy'. Bonnie Kowaliuk, Interview.

⁹⁹⁰ Heather Meath, Telephone Interview, 25 February 2008.

For Shula Edelkind, the first stage was also time-consuming, particularly determining which foods in the grocery store were acceptable: ‘I mostly got my information from person to person contact. I was on the phone during the first couple of days eight hours with a Feingold Association volunteer.’⁹⁹¹ Edelkind recalled spending ‘three hours a week at first every time I went shopping’ and added how she believed part of the difficulty parents had in the grocery store was due to brand loyalty, the idea that ‘people have emotional connections with certain brands and foods’.⁹⁹²

During the second stage of the diet, parents were encouraged to re-introduce fruits and vegetables, carefully observing whether or not certain items caused problems. This process was also challenging, but in a different way. As Heather Meath described:

That whole trial and error process of eliminating everything, bringing back certain things, finding the brands that work for you. Because they’re not the same, I don’t think, for everyone ... My son has issues with things that other people I know don’t have issues with. So working that out for yourself, it takes a while. I want to say at least six months to a year, which is an awfully long time and a big commitment.⁹⁹³

Lisa Manciewicz, however, found that the second stage of the diet was not as difficult, but this was due to the fact that her children did not react to the fruits and vegetables banned in stage one.⁹⁹⁴

For some parents, such as Colleen Davis, the perceived difficulties of the diet were such that they doubted their ability to persevere with it. Having read about the diet, Davis, whose son was being prescribed Ritalin, described how the diet

⁹⁹¹ Shula Edelkind, Interview. Most families lauded the support of FAUS in providing them with lists of acceptable foods, tips on handling Halloween and birthday parties and other advice.

⁹⁹² Ibid. Marsha Swindler also described how her weekly shopping trip expanded from a half-hour to a three and a half-hour venture. Marsha Swindler, ‘Feingold – Swindler Family’, unpublished essay written for Azusa Pacific University, 1999.

⁹⁹³ Heather Meath, Interview.

⁹⁹⁴ Lisa Manciewicz, Interview.

looked like a whole lot of work, so I didn't even bother investigating it. I thought the idea of having to feed him based on no food colouring, no flavouring and no preservatives was just going to be impossible. ... I just thought it would be a matter of I would have to feed him things he doesn't like to eat, like fruits and vegetable and whole grains and things like that. And so the medicine at that point was working for us fine. I didn't explore it further. I just thought, 'Oh those poor people on the diet!'⁹⁹⁵

If Davis, whose husband's income allowed her to stay at home with her three children, found the diet intimidating at first, and not a valid alternative to Ritalin, it is not surprising that the majority of families with hyperactive children decided against trying the diet, especially considering it was a treatment that most physicians did not support.

Single mother, Taunya Stephenson, for example, also found the diet challenging, and was unsuccessful with it at first. For Stephenson, part of the problem was her ex-husband: 'We did try the diet for the first time when he was little ... he was five I guess, but being divorced, the other party, his dad, would not participate in the diet. He would go see him often and it was just so hard.'⁹⁹⁶ While Stephenson's second attempt was more successful, many other parents were not able to persevere with it. For one parent, the diet seemed to be fairly easy at first and appeared to help her daughter. She recalled how, although the diet involved 'time consuming effort initially ... looking at the chemicals on the food and hoping that they're honestly labelled', meal preparation 'wasn't a huge change because I cooked dinners, proper dinners'.⁹⁹⁷ Within five days her daughter 'was a different child. The look she would get in her eyes when she was really frustrated was gone, it was just gone.' Despite the improvements, other problems, particularly at school, interfered with the regimen and she accepted her daughter's request to abandon the diet. Not only were the people who managed the school cafeteria unwilling to adapt their practices to help a single child, but her daughter also:

⁹⁹⁵ Colleen Davis, Interview.

⁹⁹⁶ Taunya Stevenson, Interview.

⁹⁹⁷ Anonymous, Telephone Interview, 17 January 2008.

had all these problems with people teasing her. Other people not believing that food could change her behaviour. And they would give her things and she would react. And she just didn't want to do it. And all she can remember now is that for three days all she could eat was potatoes and rice cakes.⁹⁹⁸

In contrast, some parents found that the process of changing their diet was relatively easy.

Susan Leitner's family, for example, adhered to a kosher diet and already spent time checking labels.⁹⁹⁹ Others, such as Marilee Rigg, Lora Hollins and Paula Kimball, had already adopted a diet largely free of additives. For these parents, meals prepared in the home were not as problematic as snacks, drinks and food consumed outside of the home. Marilee Rigg's son Brian, for example, reacted strongly when he ate an apple at a neighbour's house: 'A neighbour called me ... one day and said "Marilee, Brian can't eat apples, can he?" And I said, "Did you give him an apple?" She said, "Yes, and he's on the floor laughing so hard, he doesn't know what he's doing."¹⁰⁰⁰ Lora Hollins' son reacted not only to apples, but also to cider:

Once we had been to a market in ... this city that was close to us on a hot summer day, you know about thirty-five miles away from where we lived, and I had bought cider. And I'm drinking cider out of the jug and he's so thirsty and I said, 'Well here have some, what's the worst that will happen?' And so he got through a whole lot of cider and I'm telling you he was like a wild drunk Irishman. When we got home I ended up having to wrestle him and throw him down on the ground.¹⁰⁰¹

For Paula Kimball, clearing her cupboards of foods that contained additives was not particularly difficult, since she 'was Italian' and 'did all the cooking anyway', but some snacks and foods that she gave to her son as a treat were problematic.¹⁰⁰² Once she began

⁹⁹⁸ Ibid. Despite the fact that her daughter had not been on the diet for over twenty years, the anonymous mother still received information from FAUS and volunteered to be interviewed about her experiences. She continues to try to convince her daughter to consider re-attempting the Feingold diet, in order to wean herself off of the stimulant drugs that she currently is prescribed.

⁹⁹⁹ Susan Leitner, Interview.

¹⁰⁰⁰ Marilee Rigg, Interview. Rigg found out, however, that if Brian ate cooked apples or drank apple juice he was fine.

¹⁰⁰¹ Lora Hollins, Interview.

¹⁰⁰² Paula Kimball, Interview. Kimball had also given her son, Joey, organic baby food.

researching the Feingold diet, she discovered to her surprise that one of her son's favourite breakfast treats contained food colouring:

I realised I was giving him waffles in the morning, and I look on the package – the waffles are the shape of zoo animals because they market it for the kids – and there was Yellow # 5 on it ... and so I was jacking him up first thing in the morning. But I always had bought him the all natural or the pure maple syrup, so I was always doing that, but never realised that the waffle itself was what was really bad. Because I just figured, well they're marketing it for kids, it must be healthy. ... And I have learned with Joey that Yellow #5 is the worst, red comes close after, so we just avoid all artificial colours ... that just sends him – it's like he's on crack cocaine.¹⁰⁰³

Although the Feingold diet helped Joey with his behaviour at home, problems at school remained. After meeting with the school about Joey's behaviour, and subsequently pulling him from that school, Kimball found out that 'for snacks they were giving them Goldfish [a brand of cracker]. And the regular Goldfish are okay, but the extra cheesy Goldfish, or whatever it is, has the artificial colour in it. So he was having those kind of snacks during their snack-time.'¹⁰⁰⁴

In general, parents found that restricting what their children ate outside of the home was one of the most difficult aspects of the Feingold diet. While the challenges involved in controlling the food their children consumed at school influenced some parents to switch schools or, in some cases, home-school their children, others found that the schools were helpful, particularly if school officials had noted improvements in behaviour.¹⁰⁰⁵ Similarly, parents revealed differing levels of co-operation from friends and relatives. Although many parents, such as Marilee Rigg, expressed how supportive their family and friends were, others were not so fortunate.¹⁰⁰⁶ An anonymous mother, for example, recalled how her friends and

¹⁰⁰³ Ibid.

¹⁰⁰⁴ Ibid.

¹⁰⁰⁵ In order to prevent children from eating snacks at school that contained additives, for example, many parents provided bags of additive-free snacks to the school so that their child would not feel left out during 'snack-time'. Sean Corr, Interview; Kelly Anne Tooker, Interview; Paula Kimball, Interview; Colleen Davis, Interview.

¹⁰⁰⁶ Marilee Rigg, Interview.

family thought the Feingold diet was ‘ridiculous’ and that ‘food couldn’t possibly cause behaviour problems’.¹⁰⁰⁷ For other parents, one side of the family was more supportive than the other. Although Leah Hause’s parents were ‘awesome’ when it came to following the diet, her husband’s side was more troublesome, despite the fact that they had problems with certain types of food themselves:

We have a peanut allergy with a nephew in the family, and we’ve had some issues with that side of the family. They put a lot more of an ... emphasis on the peanut allergy, and haven’t really cared about my kids’ problems. We’ve had discussions about it. I’ve just gotten to the point where I’m sick of it and I’ll bring my own food ... They’ll have only pop for the kids. Or Hi-C which is basically pop ... My kids will say, ‘No thanks, we can’t have that, but Mom brought us juice boxes.’¹⁰⁰⁸

Although Feingold families faced a good deal of scepticism, such doubts could be quelled when friends or relatives witnessed the effects of a dietary violation. When in Thunder Bay, Ontario for a family visit, for example, Paula Kimball let her mother bring Joey to :

the Tim Hortons [a popular Canadian donut shop chain] ... and bought him a cinnamon bun ... and then she took him to the grocery store and we met up with an elderly lady who was a neighbour and he started spitting in her face - that was one of the things, spitting and kicking - ... and this was after a week of perfect behaviour, just very good young boy behaviour and my mother said, never again was she going to Tim Hortons.¹⁰⁰⁹

Similarly, Marta Phillips’ mother-in-law, whom Phillips thought ‘would never get it’, became convinced that the Feingold diet worked after seeing the improvement in her son, Marshall.

On the other hand, many of Phillips’ other friends and family bristled at her approach to

¹⁰⁰⁷ Anonymous, Telephone Interview, 17 January 2008.

¹⁰⁰⁸ Leah Hause, Interview.

¹⁰⁰⁹ Paula Kimball, Interview. Tim Hortons, it should be noted, is more than a mere donut shop. Founded in 1964 by Canadian hockey player, Tim Horton, who died tragically in a traffic accident a decade later, the chain sponsors the Briar, the Canadian men’s curling championship, funds Tim Hortons Children’s Foundation, which sends disadvantaged children to camp, and has a special relationship with the Canadian Armed Forces, which asked the company to open outlets at overseas bases in order to boost morale. One indication of how Tim Hortons has capitalised on its status as a symbol of national pride is evidenced in the fact that, when powerful American rival Krispy Kreme ventured into the Canadian market in 2001, it failed to dent Tim Hortons’ market share. In other words, Paula Kimball’s mother did not renounce just any donut shop, she rejected a Canadian institution. Susan Ormiston, ‘Will Heavyweight Krispy Kreme Step on Tim’s Toes?’, *Marketplace*, 20 March 2002, www.archives.cbc.ca/lifestyle/food/clips/15211/, accessed 24 February 2009.

Marshall's diet: 'They think I'm the most oversensitive, obnoxious, overbearing mother there ever was, but that's okay. I'll take that. They don't get it. But they don't live with him. They don't see how much more pleasant he is to be around.' In order to participate in family events, Phillips prepared most of the food so that she was confident that Marshall would have acceptable foods to eat, a practice many other families followed.¹⁰¹⁰

Parents also had to cope with other special occasions where food was involved, most notably birthday parties and Halloween. Often it was experiences with birthday parties that convinced parents that food additives were at the root of their child's hyperactivity. Colleen Davis became suspicious of food colours after:

a road trip to Michigan [from Virginia] for a birthday party. ... My mother provided this wonderfully decorated birthday cake ... and it was full of blue food colouring. ... Our trip home from my mom's house [the next day] was literally torture. Nikita could not sit still in his car seat, he screamed the whole time home, he complained, he thrashed, he kicked – it's a nine hour trip – and he did it for the whole trip home. And the next day he was very, very unstable and very tantrmy and that was my clue, that's what made me think that there's something about this blue food colouring. I had a friend who had mentioned to me that her son is ADHD, he's on medication, and they keep him from certain coloured food, too. So, I put those two together and I started researching food colouring and ADHD.¹⁰¹¹

Similarly, an experience following a birthday party helped to convince Susan Leitner that the Feingold diet was working for her son:

While he was on the Ritalin, I took him to a birthday party and we had been following this diet, and on the way to the birthday party, we sat in the car, he and I, and had a very intelligent conversation. He sat very nicely. It was like, 'Wow, a calm kid.' And I had made arrangements with the mom. She was going to have ice cream and cake and I had sent along a cupcake and we had heard where she was getting the ice cream and the bread and whatever and it was going to be fine. So, we're like, 'Benjamin, here's your cupcake, you can have the ice cream; don't have the cake.' And when I picked him up from the party, he was ... talking a mile a minute, a different kid, and we figured out that the mom had switched ice creams. So he was told, go ahead and have the ice cream and that point I decided okay, this was really working.¹⁰¹²

¹⁰¹⁰ Marta Phillips, Interview.

¹⁰¹¹ Colleen Davis, Interview.

¹⁰¹² Susan Leitner, Interview. Many other parents provided additive-free cupcake to eat instead of the birthday cake, but this strategy did not always work. When Heather Meath offered to make a cupcake for her son to

Leitner's experience highlights not only how parents attempted to pre-empt dietary violations at special events by preparing special food for their children, but also demonstrates how such preparation was not always sufficient. Although many parents developed creative ways in which to deal with Halloween, for example, by giving their children a toy in exchange for the candy they collected, they nevertheless had to trust that their children would not try to get at the confiscated candy. Brian Rigg, for instance, gave into temptation one Halloween:

I snuck in my candy bag and I just went crazy. I ate everything in it and got really sick. I remember my parents telling me let us inspect the candy because they're some crazy people out there; sometimes they put poison in it. So all of the sudden I thought I'd got poisoned ... I asked, 'Am I going die?' And my dad was kind of irritated with me and he said, 'If you die, you die. Go back to bed.' And I remember staying up all night crying, thinking that if I could stay awake, I wouldn't die.¹⁰¹³

This unfortunate incident notwithstanding, Rigg, as with most of the other children, was usually compliant. For Rigg, compliance equated to academic and social success. He described how, 'If I kept to my good diet, I could read, I had friends, I could control myself'; if Rigg cheated, he felt guilty and was invariably found out by his mother.¹⁰¹⁴ Similarly, Ben Leitner felt that he benefited from the Feingold diet, describing how when he cheated he 'would feel as if I didn't have as much control ... whatever would happen, I would react before I realised what I wanted to do.'¹⁰¹⁵ Guilt, however, also played a role, as Ben's mother, Susan, recalled:

I remember when he was a little older, maybe twelve or so, we went somewhere where there was three different colours of Jell-O, and he was like, 'Can I have it?' And I said, 'You're old enough for that to be a decision that you have to make yourself, but you're responsible for your behaviour if you

bring to a party, he refused because it made him feel different. Heather Meath, Interview.

¹⁰¹³ Brian Rigg, Interview.

¹⁰¹⁴ Ibid. Rigg did not feel guilty when he had a candy bar, or another banned item, 'before football games because I would go in there and just, you know, have extra energy and be all hyper because I would be hitting people in pads and beating them up'.

¹⁰¹⁵ Benjamin Leitner, Interview.

do.’ He hated that. He’d say, ‘You’re just trying to make me feel guilty,’ and, yes, I was.¹⁰¹⁶

Joshua Stevenson was also reluctant to cheat, stating that when he ‘realised that there was an improvement, it made me want to stay on it, to keep the way I was going’. It also helped that his mother ‘did a pretty good job of buying food that went with the Feingold diet, but was also good to eat’. Sean Corr similarly described how his son’s compliance was based on the recognition that it helped him to behave:

Josh, he’s really good about it. I think he really knows that if he does go off the diet, how it’ll make him act, you know, and he’s very good at resisting temptation. If somebody brings in cupcakes to school and he’s not sure if it has things in it he’ll very politely say, ‘You know, I don’t think I can have it because I don’t know what’s in it and I’m not supposed to have certain things.’ ... He makes really good decisions, probably better than what I would’ve made at that age.¹⁰¹⁷

It was also clear, however, that children could find the Feingold diet to be a struggle. It made some children, for instance, feel alienated from their peers. Kelly Anne Tooker, from Washington State, whose mother employed the Feingold diet and who followed it with her own family, recognised how:

there was always the group of parents who did things differently. I don’t think I was as aware of it as my kids are. That was more my personality as a child, I didn’t really care, but my kids have been very aware of that, that it is different. And we just recently moved ... into a different area of town, and we noticed ... they’re feeling less different than before. It really stood out where they were before. Now there’s a lot more families that are vegetarian or have different ethnic backgrounds and choose different types of food. It’s not as big of a deal. Before they really did feel it made them different.¹⁰¹⁸

¹⁰¹⁶ Susan Leitner, Interview.

¹⁰¹⁷ Sean Corr, Interview. Lora Hollins’ son, Rory, had a different reason for staying on the diet. According to Hollins, food additives not only made him ‘extremely hyperactive’ it would also make him ‘urinate on himself during the day and ... at night’, a symptom that disappeared once he started the Feingold diet. Rory was compliant, Hollins described, because ‘urinating on yourself will make you an outcast. No one wants wet pants’. Bonnie Kowaliuk also mentioned that her son’s enuresis also ceased after she put him on the diet. Bonnie Kowaliuk, Interview; Lora Hollins, Interview.

¹⁰¹⁸ Kelly Anne Tooker, Interview.

Another interviewee, who remained anonymous, also felt that the diet distinguished him as being different, describing how, ‘I felt like it set me apart – sort of like a Jew keeping Kosher, but without the holidays’.¹⁰¹⁹ This was partly because many of the foods that he had to avoid, such as pizza, candy or food at restaurants, were consumed during social occasions, in which he could not fully participate. Indeed, he recalled having primarily negative associations with the Feingold diet, stating how he resented the diet’s ‘restrictions, doubted their efficacy, and chafed under them’. The interviewee also felt bitter that, while his parents blamed his behavioural problems on dietary infractions, he was made responsible for such infractions.

The diet:

was an intriguing system for compartmentalizing. If something went wrong, it was blamed on the diet and my infractions, present, past, secret or accidental. If I was being defiant, it was the food speaking. If I got a bad grade, it was either my fault because I was ‘off’ in secret, or my fault because God was punishing me for having lied about eating something. So, the diet was mystical, sacred, mysterious, biochemical, explaining everything, explaining nothing, the will of God and the fault of society, separating me from the ‘normal person’.¹⁰²⁰

Although he felt healthier while on the diet and performed better at school, the interviewee ‘was never sure that any of this was a benefit of the diet’ and cheated on it regularly. Despite these negative feelings, he continued with the diet in college, modifying it when possible, and planned on employing it when he became a father.¹⁰²¹

Modification of the diet proved to be an effective way in which parents and their children made the Feingold diet more bearable. To a certain extent, modification was a feature of stage two of the Feingold diet, in which salicylate-laden fruits and vegetables were introduced, but many parents emphasised how they themselves were responsible for adapting the diet. As Brian Rigg explained: ‘We basically adhered to the principles of the Feingold

¹⁰¹⁹ Anonymous, Email Interview, 20 May 2008.

¹⁰²⁰ Ibid.

¹⁰²¹ Ibid. He did mention that employing the diet would lead to ‘very serious arguments’ when he and his wife have children because she comes from ‘an MSG infused culture’.

diet but we didn't follow the Feingold diet to a tee.'¹⁰²² Colleen Davis also expressed how she did not always stick to the Feingold diet, but used 'common sense and observation' to see if a certain food caused a change in her son's behaviour.¹⁰²³ While some parents modified the Feingold diet to include more foods, Bonnie Kowaliuk, who was gluten and lactose intolerant and believed that sugar caused behavioural problems, restricted additional items, added vitamins and put her son through a 'heavy metal detox' in order to cleanse his system, a process that she 'can't say enough about'.¹⁰²⁴ Other parents also highlighted how they, too, employed observation and analysis in order to alter the diet and cope at restaurants, family events and parties.¹⁰²⁵ Parents also developed strategies to cope when their children mistakenly or purposefully ate a banned substance. Paula Kimball described how if her son 'has something like that we'll just give him a bottle of water ... Drink the water, run and he'll just fall asleep ... I liken it to kind of having a hangover.'¹⁰²⁶

Similarly, parents did not rely on the Feingold diet alone to improve the behaviour and learning of their children, although they typically avoided the use of stimulant drugs, other psychological and educational strategies were also employed. Brian Rigg acknowledged the importance of the Feingold diet, for example, but he also attributed his academic success to the fact that his mother found a laboratory school associated with Texas Christian University that focussed on, among other things, re-building his damaged self esteem. As Rigg declared, 'public school would have eaten me alive and they didn't have the resources back then to deal with kids like me'.¹⁰²⁷ Rigg, as well as Joshua Stevenson, also found that sports

¹⁰²² Brian Rigg, Interview.

¹⁰²³ Colleen Davis, Interview.

¹⁰²⁴ Bonnie Kowaliuk, Interview.

¹⁰²⁵ The observational and analytical ability of many mothers, for example, might be due to the fact that many of them had either an academic background or strong interest in science. For example, Colleen Davis, Leah Hause, Lora Hollins, Paula Kimball, Lisa Manciewicz, Heather Meath, Marta Phillips and Kelly Anne Tooker all had training and work experience in either science, technology or health care.

¹⁰²⁶ Paula Kimball, Interview. Heather Meath described an almost identical strategy.

¹⁰²⁷ Brian Rigg, Interview.

and military training provided him with discipline and an outlet for aggression.¹⁰²⁸ Moreover, a number of mothers home-schooled their children, which gave them more control over what their children ate, and others used yoga, behaviour modification, music therapy and a range of parenting techniques in order to assist with behaviour and learning.¹⁰²⁹ The use of such a wide range of strategies highlights how most parents took a holistic approach to their children's development, a feature that, as demonstrated below, was mirrored in their approach to health in general.

The decisions parents made to adhere to the Feingold diet in the belief that it was in the best interest of their children's health not only required parents to be observant, analytical, patient, diligent, flexible and to defy conventional medical advice, it also demanded determination and assertiveness when it came to dealing with school authorities, medical professionals and the diet itself. As Bonnie Kowaliuk explained when her son's school tried to convince her to give him Ritalin: 'The school tried to push us that way and you have to become a very stringent advocate for your kids in that scenario and put your boundaries down as far as what you're prepared to do.'¹⁰³⁰ Similarly, Leah Hause described how she became more assertive with medical authorities after her experiences with the Feingold diet: 'I'm a pretty forceful personality to begin with, but I will definitely take a greater stance with the medical field now.'¹⁰³¹

When it came to succeeding on the diet, nearly all parents stressed that perseverance and diligence was essential, adding that families that did not try the Feingold diet or failed in the attempt often lacked such qualities. Contemplating why his mother, who was a single parent

¹⁰²⁸ Joshua Stevenson, Interview; Brian Rigg, Interview.

¹⁰²⁹ Bonnie Kowaliuk, Interview; Paula Kimball, Interview; Colleen Davis, Interview; Marta Phillips, Interview.

¹⁰³⁰ Bonnie Kowaliuk, Interview.

¹⁰³¹ Leah Hause, Interview.

for much of his childhood, succeeded with the diet while others failed, Brian Rigg stated: ‘Most people are lazy and most people are followers. They want to be told the solution to their problems. My mom is not lazy in this respect’, and added that it was easier for parents ‘to give a pill, sedate him, drug him, than have to deal with the problem’.¹⁰³² Marta Phillips also believed drug companies took advantage of parents’ desire for an easy solution: ‘its money and laziness. The public is lazy and doesn’t want to work and the drug companies want money. You put those two together and you have a nation who pops pills.’¹⁰³³

Although it seems as though parents shared a number of attributes that helped them successfully implement the Feingold diet, there was another essential factor that they held in common, namely, the steadfast belief that the diet worked. All parents, including those whose children ultimately chose not to adhere to the diet, agreed that the Feingold diet had improved their children’s behaviour. Often such beliefs were shaped by an epiphanic moment. After attempting a wide range of treatments for her son’s behaviour, Shula Edelkind, for instance:

talked to my child who was willing, and my husband was willing to cooperate, and we started the diet. And in four days, I’ll never forget, he walked into the kitchen and said, ‘Mom, I can’t find my other sock.’ And I just about fell to the floor because this is not something this child could’ve ever said. He would either have been hysterical because he wanted his other sock or he would’ve forgotten that he needed another sock... From that point he was quite normal emotionally.¹⁰³⁴

Colleen Davis recalled how her son’s behaviour also improved markedly after:

about four days ... and then he was just a dramatically different child. He wasn’t crying as much; he wasn’t as wild. He was sleeping through the night much better ... I could look him in the eye and talk to him and reason with him and explain things to him.¹⁰³⁵

¹⁰³² Brian Rigg, Interview.

¹⁰³³ Marta Phillips, Interview.

¹⁰³⁴ Shula Edelkind, Interview.

¹⁰³⁵ Colleen Davis, Interview.

For Leah Hause, improvements in her son's behaviour also occurred quickly and were not limited to behaviour: 'I did not expect it to have the impact on behaviour that it did. I expected it to take care of rashes. I couldn't believe how much it helped with the eczema and ... the digestive system issues. I was very amazed.'¹⁰³⁶

Other parents had to wait longer for improvements, but were equally impressed when they eventually occurred. Taunya Stevenson was told by FAUS that:

if they'd been on a lot of medicine it would take six weeks ... and I am not lying when I tell you that it was six weeks to the day ... night and day difference. Everybody noticed. ... He could sit and have a conversation without being all over the room. He would be compliant. You could ask him to do this or that and he'd just say ok and he would just do it without this huge ordeal. The teachers, I didn't get phone calls and notes and all of that just calmed down so much. There weren't problems like there were before. His aggressiveness was a big change. His aggressiveness and compliance were some of the biggest changes I saw.¹⁰³⁷

Lisa Manciewicz, whose son had previously required some special education measures, found that his teachers were also impressed by his academic improvement: 'he was out of the learning centre pretty much within six months. His teacher came to me and said, "What is going on with this? He doesn't need us anymore. He's thriving and succeeding all on his own." I said I simply changed his food.'¹⁰³⁸

As discussed in Chapter 9, one of the chief suspicions researchers had of the Feingold diet was that its effects were only a placebo, largely due to the increased attention given to the child. Interviews with a number of the researchers indicated that many continue to stress the role of placebo in creating the illusion that the Feingold diet worked.¹⁰³⁹ When parents were asked about this possibility, however, they were resolute that this was not the case. Shirley

Fadden, for example, declared that:

¹⁰³⁶ Leah Hause, Interview.

¹⁰³⁷ Taunya Stevenson, Interview.

¹⁰³⁸ Lisa Manciewicz, Interview.

¹⁰³⁹ For example, C. Keith Conners, Interview; Esther H. Wender, Telephone Interview, 17 February 2009.

I know this is not a placebo effect. For example last year he snuck money to school and bought school lunch (pizza). I did not know this. He ran in the house like a tornado and I just thought, ‘Wow I haven’t seen this in a long time.’ Finally my daughter told me he had bought lunch. He was hyper the entire day and woke up the next morning still hyper. He came off the school bus that day and he was fine. He does not sneak anymore.¹⁰⁴⁰

For other parents, such as Sean Corr, the times when his son went off his diet were enough to reaffirm his belief in the diet and undermine the notion that it was only a placebo: ‘At first you don’t notice the difference in him, you know, until he goes off his diet ... and then you’re like, “Holy cow! Now I remember why we keep him on this diet.”’ Regardless of what mainstream medicine claimed about the Feingold diet, the Corrs, and the other parents, had become ‘believers’.¹⁰⁴¹ For them the diet worked.

Conclusion

It is difficult to listen to interviews of Feingold families and not feel inclined to believe, as they did, that the Feingold diet had an enormously positive impact on their children’s lives. But as historical evidence, oral history interviews must be viewed as critically as any other source. Put another way, just as the few dozen trials of the Feingold diet failed to confirm conclusively that Feingold’s theory was invalid, the experiences of a few dozen Feingold families did not prove that it worked. Given the small sample size, it is possible that, as many of Feingold’s critics suggested, the children who responded to the Feingold diet represented an exceptional, self-selecting and miniscule percentage of the millions of children diagnosed with hyperactivity. Although it would be remarkable for the placebo effect to have caused the improvements in behaviour in all of the families interviewed, especially given the time period covered in many cases, most families did employ other interventions in addition to the Feingold diet that might have resulted in improved behaviour.

¹⁰⁴⁰ Shirley Fadden, Interview. Other parents also disagreed with the notion that the changes in behaviour they witnessed, often lasting over a period of decades, were merely placebo. Anonymous, Email Interview, 26 January 2008; Lynn Murphy, Interview.

¹⁰⁴¹ Sean Corr, Interview.

Most importantly, as Chapters 2 and 6 contend, medical and popular conceptualisations of hyperactivity have been influenced not only by trends in medical theory and technology, but also by educational, political, cultural, economic and demographic factors. Hyperactivity was, and continues to be, a disorder characterised by a mismatch of behaviours and social circumstances; behaviour deemed to be pathological in one context may be seen as beneficial in another. This is not to discount the experiences of Feingold families, many of whom were deeply troubled by their children's behaviour, but it nevertheless highlights how a variety of factors influenced such behaviour at any given time or place.

What is safer to say, however, is that this history of Feingold families reinforces Roy Porter's contention that physicians have not always been the primary agents of health care, that people sought their own cures before seeking medical advice and, when physicians were found wanting, they looked elsewhere.¹⁰⁴² In the case of Feingold families, one could go even further: parents, particularly mothers, became the medical experts regarding many aspects of the health of their children. When conventional medical solutions were unacceptable, parents conducted research, weighed the available evidence, and then experimented with the Feingold diet, observed its effects, modified it according to their requirements and made the decision to persevere with it. Although they believed that physicians were required for some interventions, parents nevertheless took responsibility for most aspects of their children's health. As Lisa Manciewicz explained

I think that there is a time and place for everything. ... My son would have died ten years ago if they didn't have the drugs and technology to perform his heart surgery. However, that doesn't mean that every time he has an ear infection or something like that they should constantly be putting him on antibiotics ... It's a band aid. They don't want to take the time to find out what's really bothering this child. Let's drug him. And then you have all of these pharmaceutical companies, they're making a fortune, and the food companies the same thing. The meat market, they pump the cow with steroids to get another twenty extra

¹⁰⁴² Porter, 'The Patient's View', 194.

steaks out of it. Well who eats that hormone? We do. Why are little girls getting breasts at ten years old and getting their period at ten?¹⁰⁴³

If parents can be seen as experts in certain aspects of their children's health, then it seems clear that medical historians should increasingly regard parents as an essential and important feature in the history of medical debates concerning children. Moreover, the history of the Feingold families suggests that parent and patient accounts could have played a much larger role in informing the debate about the Feingold diet itself.¹⁰⁴⁴ Patient and parent experiences, in conjunction with the results of the double-blind trials, might not have resolved the debate, but would likely have encouraged researchers to continue exploring the link between food additives and behaviour, perhaps in more innovative ways, such as a longitudinal study of Feingold families.¹⁰⁴⁵ Instead, the experiences of families were largely dismissed as unhelpful anecdotes that muddied the evidence emerging from the trials. Given the litany of problems that plagued these trials, it is ironic how unimportant the parental accounts were seen to be by most researchers.

Despite being excluded from official debates, parents were nevertheless able to keep Feingold's idea alive when most physicians had rejected it. Through thousands of regional Feingold Associations, which would be later centralised as FAUS, and also through informal networking, a small, but significant, number of parents whose children had been diagnosed with hyperactivity continued to discover the Feingold diet. Indeed, most parents interviewed,

¹⁰⁴³ Lisa Manciewicz, Interview. Parents tended to express more anger at pharmaceutical companies and the food industry than they did at mainstream physicians for downplaying Feingold's theory. Physicians, most thought, simply lacked a more holistic education, particularly when it came to nutrition. As Marilee Rigg described: 'We do have doctors that are really interested in nutrition, but the majority of them have not had a lot of training in medical school on nutrition and I think that's a serious problem.' Marilee Rigg, Interview.

¹⁰⁴⁴ Or as Rima Apple argues, there is a 'need to ensure that scientific and medical professionals and mothers have the resources necessary to learn from each other'. Apple, *Perfect Motherhood*, 167.

¹⁰⁴⁵ Similar approaches have been used in assessing the efficacy of other hyperactivity treatments. See: Peter S. Jenson, Stephen P. Hinshaw, James M. Swanson, Laurence L. Greenhill, C. Keith Conners, L. Eugene Arnold, Howard B. Abikoff, Glen Elliot, Lily Hechtman, Betsy Hoza, John S. March, Jeffrey H. Newcorn, Joanne B. Severe, Benedetto Vitiello, Karen Wells and Timothy Wigal, 'Findings from the NIMH Multimodal Treatment Study of ADHD (MTA): Implications and Applications for Primary Care Providers', *Developmental and Behavioral Pediatrics* 22 (2001), 60-73.

as well as their grown-up children, recommended the diet to others, sharing not only their success stories, but also the challenges of avoiding food additives and how they overcame them. By continuing to employ and promote the Feingold diet, parents have, as the final chapter demonstrates, encouraged a handful of researchers in the twenty-first century to consider Feingold's hypothesis once again.

Chapter 11

Conclusion

The purpose of this thesis has been to analyse how physicians, the media and the public decided whether or not the Feingold diet was a valid treatment for hyperactivity. It has examined the context in which Feingold's theory emerged, its dissemination from San Francisco to the rest of the United States and beyond, and how it was received and assessed by medical and lay communities. Although double-blind trials were conducted to test Feingold's hypothesis, and have been the basis upon which most medical opinions about the Feingold diet have rested, I have argued that a wide range of ideological, political and socio-economic factors were substantially more important in determining how the Feingold diet was understood, discussed and evaluated.

From the manner in which Feingold depicted the origins of his theory to the reasons why it became a popular phenomenon and to the way in which debates about his hypothesis were resolved, the history of the Feingold diet demonstrates how novel medical ideas have had to serve the interests of numerous parties. Physicians, politicians, industries, the media and patients and their families conceptualised the Feingold diet in disparate ways and for different reasons, and this complicated the debates that Feingold's idea generated. While the media saw the Feingold diet as an exciting story that would sell newspapers, the food and chemical industries saw it as a threat to how it conducted business. Although Feingold families found that the diet gave them hope, many physicians and medical researchers viewed it with suspicion, and believed that it discouraged families from accessing conventional treatments for hyperactivity. The history of the Feingold diet suggests that, during the late twentieth century in the United States, medical knowledge was not a steadily growing body of

unquestioned information and practices that was universally accepted, but instead a mutable and fluid series of explanations and understandings that vied with each other for legitimacy. The reason one idea appealed to one particular party could have as much to do with politics, ideology or economics as it had to do with the weight of scientific evidence that supported it or the way in which it helped patients.

An undercurrent to this thesis has been the use of history as a way in which to analyse and assess the outcomes of medical debates. Historians bring critical and contextual perspective to understanding why certain medical theories achieved legitimacy and others did not and, as such, are in a position to help inform public health policy. While some historians have begun to address such issues with reference to the histories of immunology and psychiatry, such accounts have often failed to deconstruct how scientific knowledge is made authoritative or, conversely, have applied social theories too bluntly in an attempt to explain the uptake of particular medical ideas. This thesis has contended that the development of psychiatric and immunological knowledge has been a considerably more subtle, complex and often contradictory process, and one that reveals as much about the elements of society involved as it does about the science. It is hoped that the history of the Feingold diet has provided not only a case study about an unusual explanation for hyperactivity, but has also shown how changes in how Americans understood and dealt with mental illness and allergy during the twentieth century reflected broader debates about the education of children, the testing of scientific ideas, the use of psychoactive drugs, the presence of chemicals in the food supply and the role of parents in determining which medical treatments were best for their children. If this is true it suggests that, as society evolves, attitudes to medical notions once presumed to be incorrect can also change.

In 2004 an article appeared in the *Archives of Disease in Childhood* which put Feingold's theory to the test yet again. Bowing to public pressure, the British Food Standards Agency (FSA) had issued a call for proposals to test whether the behaviour of children in the general population was affected by food additives.¹⁰⁴⁶ The research group that was awarded the funding was from the University of Southampton and was led by psychologist Jim Stevenson. The group designed a double-blind trial which tested the behavioural responses of 277 three-year-old children from the Isle of Wight to challenges of artificial food colourings and the preservative sodium benzoate.¹⁰⁴⁷ Although formal testing did not confirm that the additive-free diet reduced hyperactivity, parental rating scales did, and the researchers concluded that 'significant changes in children's hyperactive behaviour could be produced by the removal of artificial colourings and sodium benzoate from their diet' and that 'benefit would accrue for all children if artificial food colours and benzoate preservatives were removed from their diet'.¹⁰⁴⁸ Two letters to the editor which appeared on the journal's website soon after, however, indicated that, thirty years after Feingold had published *Why Your Child is Hyperactive*, his idea continued to divide opinion.

The first letter, from a physician, who worked in private medical practice, was enthusiastic:

¹⁰⁴⁶ Jim Stevenson, Telephone Interview, 16 March 2009.

¹⁰⁴⁷ B. Bateman, J. O. Warner, E. Hutchinson, T. Dean, P. Rowlandson, C. Grant, J. Grundy, C. Fitzgerald and J. Stephenson, 'The Effects of a Double-Blind, Placebo Controlled, Artificial Food Colourings and Benzoate Preservative Challenge on Hyperactivity in a General Population Sample of Preschool Children', *Archives of Disease in Childhood* 89 (2004), 506-11. The team's focus on children from the Isle of Wight is interesting because children from the Isle of Wight were also studied by prominent British child psychiatrist Sir Michael Rutter in one of the first epidemiological studies of childhood mental health during the mid-1960s. Rutter later compared the rates of mental illness of children on the Isle of Wight with that of children from inner London in 1970. He found that the London children had twice the rate of mental illness as those from the Isle of Wight, and posited that higher levels of stress affecting not only the children, but also their parents, were responsible for the higher rates. Such findings accorded with the social psychiatry prominent during the 1960s, and reflect how social psychiatry had a greater and more enduring impact in Britain than it did in the United States. Michael Rutter, J. Tizard, W. Yule, P. Graham and K. Whitmore, 'Research Report: Isle of Wight Studies, 1964-1974', *Psychological Medicine* 6 (1976), 313-32; Carl I. Cohen, Joel S. Feiner, Charles Huffine, H. Steven Moffic, Kenneth S. Thompson, 'The Future of Community Psychiatry', *Community Mental Health Journal* 39 (2003), 459-71, at p. 460; Michael Rutter, 'Isle of Wight Revisited: Twenty-Five Years of Child Psychiatric Epidemiology' in Stella Chess and Margaret E. Hertzog (eds.), *Annual Progress in Child Psychiatry and Child Development* (New York: Psychology Press, 1990), 131-79, at p. 148.

¹⁰⁴⁸ Bateman, *et al.*, 'Effects of a Double-Blind', 510-1.

I remember the days of cramming for exams, working part-time and checking off the remaining days to the end of the torture in my diary. I am talking about the seventies, when petrol crises alternated with political disasters like the Nixon Gate. It was then that we first heard of Dr Feingold's revolutionary findings: Apparently, colourings and other chemicals in food and environment could cause behaviour problems and learning difficulties. A very sexy and down to earth psychology professor persuaded many of us to forego the Hostess Twinkies, the Hot Dogs and the beautifully coloured licorice twists, fig Newtons and Oreo cookies. Of course, being mature beyond our years, we aimed to please and soon found other staples. And there was no doubt about it, the therapy was effective. Having only read the abstract I can't say whether credit was given where credit is due but suffice it to say that Feingold was ahead of his time. May he rest snugly.¹⁰⁴⁹

The other letter, written by a medical professor at the University of North Carolina, was less enthusiastic:

Having been an interested observer to the Feingold Hypothesis many years ago, I was startled to see *it rise from the dead* (highlighted in many medical excerpting services). I eagerly downloaded this article, and shortly thereafter, my thoughts could be paraphrased in a well-done American advertisement: 'Where's the meat?' Figure 3 screamed at me one obvious conclusion: 'Parents are sensitive to knowing something was to be changed in their child's environment.' The withdrawal phase, placebo phase, and challenge phase ALL seemed to cause identical responses in both experimental orders. Imagine if the two groups (e.g., placebo-challenge vs challenge-placebo) had instead been a repeat experiment done at a different time. For a clinical study, the obvious conclusion was 'wow, really tight, repeatable findings.' Instead, some manner of statistics has overwhelmed common sense, leading to wide publicity of a 'toxic effect.' Most people will never read this manuscript, and the reviewers and editors owed us careful thought before opening up this Pandora's Box. The field will not Find-Gold with Feingold.¹⁰⁵⁰

The two letters suggested that the dilemma at the heart of debates about the Feingold diet has persisted: although Feingold's hypothesis seemed sensible to many people, and although his diet appeared to work, it was nevertheless difficult to prove, largely because its effects could be attributed to placebo effect, rather than dietary change.

¹⁰⁴⁹ Herbert H. Nehrlich, 'Feingold Revisited and Acknowledged', *Archives of Disease in Childhood* 89 (2004), www.adc.bmj.com/login.ezproxy.library.ualberta.ca/cgi/eletters/89/6/506#927, accessed 6 March 2009.

¹⁰⁵⁰ Italics and uppercase in original. Richard B. Mailman, 'Where's the Effect?', *Archives of Disease in Childhood* 89 (2004), www.adc.bmj.com/login.ezproxy.library.ualberta.ca/cgi/eletters/89/6/506#927, accessed 6 March 2009.

Although the second letter questioned whether the trial was actually blind, the editorial that accompanied the article described it as ‘ameticulously performed, double-blind, placebo controlled trial’, stated that it was ‘unlikely’ that parents detected which diet their children were consuming, and ‘congratulated’ the authors ‘for tackling a complicated subject, in a rigorous manner’.¹⁰⁵¹ The editorial also addressed the issue of whether the parental rating scales should be considered more valid than formal tests of hyperactivity. According to the study’s authors:

Parental ratings might be more sensitive to changes in behaviour in that parents experience their child’s behaviour over a longer period of time, in more varied settings and under less optimal conditions. The tests conducted in clinic are liked by the majority of children who see them as an entertaining game; they are given when the children are optimally alert and engaged. In contrast, parents will observe the child’s behaviour when they are competing with siblings for attention; at times when the child is hungry or tired; when the child has less devoted attention from one adult; when the child is interacting with other children; or in a constraining setting such as on public transport or in a supermarket queue.¹⁰⁵²

Although the editorial itself was hesitant to endorse unconditionally the validity of parental ratings, it nonetheless implied that physicians should reconsider how they perceived parental observations of childhood behaviour, and acknowledged that the study would ‘fuel the debate that there are environmental causes of hyperactivity, and that prior to medicating children we need to aggressively eliminate them [the environmental causes of hyperactivity]’.¹⁰⁵³

Three years later, Stevenson led another study that compared the effect of an additive-free diet on 153 three-year-old and 144 eight and nine-year-old children. Their findings, which were published in the *Lancet*, provided ‘strong support for the case that food additives exacerbate hyperactive behaviours ... in children at least up to middle childhood’ and showed that such increases were ‘not just seen in children with extreme hyperactivity (ie, ADHD) but

¹⁰⁵¹ Howard Bauchner, ‘Food Colourings and Benzoate Preservatives – Do They Change Behaviour?’ *Archives of Disease in Childhood* 84 (2004), 499.

¹⁰⁵² Batemen, *et al*, ‘Effects of A Double-blind’, 510.

¹⁰⁵³ Bauchner, ‘Food Colourings’, 499.

also can be seen in the general population and across the range of severities of hyperactivity'.¹⁰⁵⁴ The authors added that the 'implications of these results for the regulation of food additive use could be substantial', and the FSA proceeded to revise their advice to parents about the safety of food colours.¹⁰⁵⁵

Unlike the 2004 study, which attracted little media attention, the 2007 trial generated a flood of reports in print and on television, radio and the internet, much to the surprise of Jim Stevenson.¹⁰⁵⁶ It also garnered attention from the AAP which published a summary of the research results in *AAP Grand Rounds*, a digest of the paediatric research most relevant to clinicians. The commentary, written by Alison Schonwald, stated that the researchers' findings gave 'practitioners ... a reasonable option to offer parents'.¹⁰⁵⁷ Moreover, the editor's note that followed the commentary stated not only that the trial 'was a carefully conducted study in which the investigators went to great lengths to eliminate bias and rigorously measure outcomes', but also that 'the overall findings of the study are clear and

¹⁰⁵⁴ Donna McCann, Angelina Barrett, Alison Cooper, Debbie Crumpler, Lindy Dalen, Kate Grimshaw, Elizabeth Kitchin, Kris Lok, Lucy Porteous, Emily Prince, Edmund Sonuga-Barke, John O. Warner, Jim Stevenson, 'Food Additives and Hyperactive Behaviour in 3-Year-Old and 8/9-Year-Old Children in the Community: A Randomised, Double-Blinded, Placebo-Controlled Trial', *Lancet* 370 (2007), 1560-7, at p. 1566.

¹⁰⁵⁵ Food Standards Agency, 'Agency Revises Advice on Certain Artificial Colours', 11 September 2007, www.food.gov.uk/news/newsarchive/2007/sep/foodcolours, accessed 20 March 2009; McCann, *et al.*, 'Food Additives and Hyperactivity', 1566. When interviewed, Jim Stevenson described how the FSA could not go further in curtailing the use of such additives because of EU restrictions. Jim Stevenson.

¹⁰⁵⁶ For example, David Andreatta, 'Food Additives Found to Fuel Hyperactivity', *Globe and Mail*, 6 September 2007, www.theglobeandmail.com/servlet/story/RTGAM.20070906.whyperkids06/BNStory/specialScienceandHealth/home, accessed 9 March 2009; Anonymous, 'Parents Warned of Additives Link', 6 September 2007, www.news.bbc.co.uk/1/hi/health/6979976.stm, accessed 9 March 2009; Valerie Elliot, 'Food Alert as Every Additive Comes Under Suspicion', *The Times*, 6 September 2007, www.timesonline.co.uk/tol/news/uk/health/article2395623.ece, accessed 9 March 2009; Krishnan Guru-Murthy, *Channel 4 News*, 6 September 2007. Journalistic interest was matched by that of the general public. Andrew Wadge, the FSA's chief scientist, for example, commented on the FSA website that he could not 'think of another topic that's generated so much feedback so quickly, expressed with a strength of feeling that I don't think we've seen on the blog before'. Stevenson suspected that much of the increased interest was due to the fact that the study was published in the *Lancet*. Jim Stevenson; Andrew Wadge, 'Colours and Hyperactivity', www.fsascience.net/2007/09/06/colours_and_hyperactivity, accessed 9 March 2009.

¹⁰⁵⁷ Alison Schonwald, 'ADHD and Food Additives Revisited', *AAP Grand Rounds* 19 (2008), 17.

require that even we skeptics, who have long doubted parental claims of the effects of various foods on the behavior of their children, admit we might have been wrong'.¹⁰⁵⁸

Thirty-five years after Feingold presented his research to the AMA and failed in his attempt to publish his findings in *JAMA*, this statement represented a major reversal in how at least one major American medical association perceived the connection between food additives and hyperactivity. Despite the AAP's acknowledgement that the 2007 study lent support to Feingold's hypothesis, however, it would be a mistake to attribute their admission of being mistaken to the results of the study alone. Both of the Stevenson studies certainly had fewer methodological problems and involved a larger number of participants than nearly all of the trials that occurred during the 1970s and early 1980s. But most studies that emerged during the late 1980s and 1990s, while they failed to attract much media or medical attention, also tended to be better designed and yielded results that supported Feingold's theory. Bonnie Kaplan's trial, described in Chapter 9, was one such trial, but there were a handful of others that provided supportive evidence.¹⁰⁵⁹

¹⁰⁵⁸ Anonymous, 'Editor's Note', *AAP Grand Rounds* 19 (2008), 17. Swiss paediatricians, Phillippe Eigenmann and Charles Haenggeli, who had criticised the group's 2004 research, stating that they 'strongly believe that unnecessary diets should not be instituted for hyperactivity', also adjusted their views following the 2007 *Lancet* article. While they cautioned that the results would not be applicable to all hyperactive children, they nonetheless admitted that physicians 'taking care of children with hyperactivity could advise parents who wish to do so to start an elimination diet of artificial colourings and additives'. Philippe A. Eigenmann and Charles A. Haenggeli, 'Food Colourings and Preservatives: Allergy and Hyperactivity', *Lancet* 364 (2004), 823-4; Philippe A. Eigenmann and Charles A. Haenggeli, 'Food Colourings, Preservatives and Hyperactivity', *Lancet* 370 (2007), 1524-5, at p. 1525.

¹⁰⁵⁹ For example, J. Egger, C. M. Carter, P. J. Graham, D. Gumley and J. F. Soothill, 'Controlled Trial of Oligoantigenic Treatment in the Hyperkinetic Syndrome', *Lancet* 325 (1985), 540-5; Bonnie J. Kaplan, Jane McNicol, Richard A. Conte, H. K. Moghadam, 'Dietary Replacement in Preschool-Aged Hyperactive Boys', *Pediatrics* 83 (1989), 7-17; K. S. Rowe, 'Synthetic Food Colourings and "Hyperactivity": A Double-Blind Crossover Study', *Australian Paediatric Journal* 24 (1988), 143-7; I. Pollock, J.O. Warner, 'Effects of Artificial Food Colours on Childhood Behaviour', *Archives of Disease in Childhood* 65 (1990), 74-7; M. Boris and F. S. Mandel, 'Foods and Additives Are Common Causes of the Attention Deficit Hyperactive Disorder in Children', *Annals of Allergy* 72 (1994), 462-8; K. S. Rowe and K. J. Rowe, 'Synthetic Food Coloring and Behavior: A Dose Response Effect in a Double-Blind, Placebo-Controlled, Repeated-Measures Study', *Journal of Pediatrics* 125, (1994), 691-8.

Moreover, despite having addressed some of the methodological problems, the 2004 and 2007 trials were not flawless, and critics questioned how the researchers defined hyperactivity, controlled their trials and interpreted their results.¹⁰⁶⁰ The AAP's reversal, as well as the increase in media interest, had less to do with the emergence of a convincing, decisive study as it did with cultural, technological and political developments that made the Feingold diet appear, once again, to be a viable alternative to conventional treatments for hyperactivity. In particular, many dynamics, including concern about the health of the food supply, growing consumer wariness about drugs and new information technologies had intervened to re-invigorate interest in the Feingold diet. Furthermore, the centre for research and debate regarding Feingold's hypothesis had shifted from the United States to other jurisdictions, most notably, Britain. In order to conclude this examination of the history of the Feingold diet, this concluding chapter analyses why Feingold's theory has experienced a renaissance, particularly, but not exclusively, in Britain, and outlines how the history of the Feingold diet can help to inform future debates about controversial medical ideas.

One compelling aspect of the media storm that followed the results of Stevenson's 2007 study was that the majority of media interest emanated from Britain, rather than the United States. This tendency was opposite to the media response that accompanied Feingold's research during the 1970s, and indicated how medical and media interest in the Feingold diet had relocated from one side of the Atlantic to the other. While American families continued to discover and attempt the Feingold diet, American researchers were no longer leading investigations into Feingold's hypothesis. In fact, this trend had begun during the mid-1980s,

¹⁰⁶⁰ For example, Mailman, 'Where's the Effect'; Eigenmann and Haenggeli, 'Food Colourings', 823; Maggie Fox, 'Food Additives May Cause Hyperactivity: Study', 5 September 2007, www.reuters.com/article/healthNews/idUSN0520103220070905, accessed 7 September 2007; Andreatta, 'Food Additives'; Julian Hunt quoted in Anonymous, 'Food Watchdog Condemned for 'Totally Inadequate' Response to Harmful Food Additives', *This is London*, 6 September 2007, www.thisislondon.co.uk/news/article-23411169-details/Parents+warned:+additives+in+food+DO+harm+our+children/article.do, accessed 13 March 2009; C. Keith Connors, Interview; Esther Wender, Interview.

following Feingold's death, as most of the small number of trials conducted during the late 1980s and 1990s were British, along with a few Australian and Canadian studies.¹⁰⁶¹

To a degree, growing interest in the Feingold diet in Britain paralleled increased British interest in hyperactivity more generally. Although hyperactivity was the most common American childhood mental health issue by the late 1960s, it took longer for the disorder to become widespread in Britain. As described in Chapter Two, one of the reasons why hyperactivity became predominant in American child psychiatry during the late 1950s involved how it was described and defined by Maurice Laufer, Eric Denhoff and Gerald Solomons in 1957. 'Hyperkinetic Impulse Disorder', the term they coined, which was truncated to hyperkinesis or, more commonly, hyperactivity, was a broad category that encapsulated a wide range of childhood behaviours.¹⁰⁶² British physicians and educators, however, were reluctant to embrace the term and, instead, employed a range of other labels to describe children with behavioural and learning problems. While British schoolchildren were often described by educators as 'maladjusted' or 'medium educational subnormal', British psychiatrists might diagnose them with 'conduct disorder', 'school phobia', 'emotional disorder' or even 'autism'.¹⁰⁶³ When British psychiatrists, such as Sir Michael Rutter, did diagnose children with hyperactivity, the symptoms were more severe than those described in diagnoses of hyperactivity made in North America.¹⁰⁶⁴

Although British psychiatrists seemed reluctant to emulate their American colleagues in diagnosing millions of children with hyperactivity and prescribing amphetamines for treating

¹⁰⁶¹ For instance, Egger, *et al*, 'Controlled Trial'; Boris and Mandel, 'Foods and Additives'; Rowe, 'Synthetic Food Colourings'; Kaplan, *et al*, 'Dietary Replacement'; Pollock and Warner, 'Effects of Artificial Food'; Rowe and Rowe, 'Synthetic Food Coloring'.

¹⁰⁶² M. W. Laufer, E. Denhoff and G. Solomons, 'Hyperkinetic Impulse Disorder in Children's Behavior Problems', *Psychosomatic Medicine* 19 (1957), 38-49.

¹⁰⁶³ Steven Box, 'Preface', in Peter Schrag and Diane Divoky, *The Myth of the Hyperactive Child: And Other Means of Child Control* (New York: Penguin Books, [1975] 1982), 7-30, at p. 17.

¹⁰⁶⁴ Anonymous, 'Minimal Brain Dysfunction', *Lancet* 302 (1973), 487-8; Mark A. Stewart and B. H. Burne, 'Minimal Brain Dysfunction', *Lancet* 302 (1973), 852.

the disorder, confusion existed about which approach was preferable. A 1973 editorial in the *Lancet*, for instance, asked: ‘Are the Americans ahead of the British, or behind them, or do their children’s brains dysfunction in such an ostentatiously exotic transatlantic fashion that they require drug therapy?’¹⁰⁶⁵ By 1978, British physicians were still wary of the American approach:

hyperactivity ought to mean nothing more than increased (or excessive) activity, but all too often the word is used to describe a neurobehavioural disturbance which should be treated with potent drugs. In the U.S.A. at least 5% of all normal schoolchildren are thought to be victims of the hyperactivity syndrome, and many of them are given treatment. The position in the U.K., though less alarming, is not negligible and could become worse. Fortunately, over the years Rutter and his colleagues have been painstakingly studying behavioural symptomatology and now conclude that ‘here is *no* evidence for the validity of a broader concept of hyperkinetic syndrome’.¹⁰⁶⁶

Eight years later, however, British physicians were less confident about their proclivity to disregard the American approach to defining and treating hyperactivity. Another *Lancet* editorial described how ‘British paediatricians, family practitioners and child psychiatrists are far less ready than their colleagues in the USA to diagnose and treat a syndrome of hyperactivity’, but then proceeded to warn that ‘severe and pervasive hyperactivity is a risk factor and can handicap social development’ and that ‘British medicine and education will need to make its modification a higher priority.’¹⁰⁶⁷ Such warnings were heeded, as amphetamine prescriptions in Britain rose from 183 000 in 1991 to 1.58 million in 1995.¹⁰⁶⁸ Despite the increase, British prescription rates remained much lower than in North America

¹⁰⁶⁵ Anonymous, ‘Minimal Brain Dysfunction’, 488.

¹⁰⁶⁶ Italics in original. Anonymous, ‘Hyperactivity’, *Lancet* 312 (1978), 561.

¹⁰⁶⁷ Anonymous, ‘Does Hyperactivity Matter?’, *Lancet* 327 (1986), 73-4. The first mention about hyperactivity in the *Lancet* did not appear until 1970 and was a letter to the editor, along with a description of research, written by American psychiatrists. The only response to the letter was also by an American psychiatrist. J. L. Rapoport, I. T. Lott, D. F. Alexander, A. U. Abramson, ‘Urinary Noradrenaline and Playroom Behaviour in Hyperactive Children’, *Lancet* 296 (1970), 1141; Mark. A. Stewart, ‘Urinary Noradrenaline and Playroom Behaviour in Hyperactive Children’, *Lancet* 297 (1971), 140.

¹⁰⁶⁸ Morris Zwi, Paul Ramchandani and Carol Joughin, ‘Evidence and Belief in ADHD’, *BMJ* 321 (2000), 975-6.

and British physicians continued to debate whether or not the disorder was underdiagnosed.¹⁰⁶⁹

Perhaps because of their reluctance to embrace an American-style biomedical approach to hyperactivity, British physicians came to understand hyperactivity in a more pluralistic manner. This was reflected not only in Rutter's holistic approach to childhood mental illness, and his work on its social aetiologies in particular, but also in the fact that many of the first articles written about hyperactivity in British medical journals focussed on alternative explanations for the disorder, in particular, theories about lead exposure and, indeed, about food additives.¹⁰⁷⁰ Although there were exceptions, particularly the commentaries of Thomas Jukes, who worked for a chemical company and had a history of supporting the use of food additives, most editorials about the Feingold diet were supportive, and seemed to be more concerned about how to define hyperactivity than the possibility that chemicals in food could cause behavioural problems.¹⁰⁷¹ British parents were also eager to consider the link between additives and hyperactivity, and looked to organisations such as the Hyperactive Children's Support Group, founded in the late 1970s by Sally Bunday for assistance in planning an additive-free diet.¹⁰⁷²

Increased concern about and a pluralistic approach to hyperactivity were not the only factors, however, that contributed to British interest in the Feingold diet. Many of the developments

¹⁰⁶⁹ Geoffrey D. Kewley, 'Personal Paper: Attention Deficit Hyperactivity Disorder is Underdiagnosed and Undertreated in Britain', *BMJ* 316 (1998), 1594-6; Eileen Orford, 'Commentary: Diagnosis Needs Tightening', *BMJ* 316 (1998), 1594-6.

¹⁰⁷⁰ David, Clark, and Voeller, 'Lead and Hyperactivity'; Anonymous, 'Subclinical Lead Poisoning', *Lancet* 301 (1973), 87; Anonymous, 'Feingold's Regimen'; Anonymous, 'Food Additives and Hyperactivity'; Egger, *et al.*, 'Controlled Trial'; Ann Swain, Velencia Soutter, Robert Loblay and A. Stewart Truswell, 'Salicylates, Oligoantigenic Diets, and Behaviour', *Lancet* 326 (1985), 41-2.

¹⁰⁷¹ Anonymous, 'Feingold's Regimen'; Anonymous, 'Food Additives and Hyperactivity'; Thomas H. Jukes, 'Language in Action', *Nature* 264 (1976), 602; Thomas H. Jukes, 'Friedrich Wöhler RIP', *Nature* 273 (1978), 421.

¹⁰⁷² The Hyperactive Children's Support Group also published a collection of success stories entitled *The Proof of the Pudding*. Hyperactive Children's Support Group, 'Our Publications', www.hacsg.org.uk/HACSG%20PUBLICATIONS.htm, accessed 16 March 2009.

that made the Feingold diet popular in United States during the early 1970s, particularly concern about food safety and fears about psychiatric drugs, became prevalent for different reasons in Britain during the 1990s and 2000s, and encouraged both British parents and medical researchers to consider the link between food additives and hyperactivity. Moreover, the British food industry and government, unlike their counterparts in the United States, believing that such a link might be valid, took pro-active steps to reduce the amount of additives in food, especially foods commonly consumed by children.

Although the British organic food movement dated back to the 1930s and attracted both right and left wing adherents, a number of specific events related to the public's perception of the food and pharmaceutical industries during the 1990s and 2000s created a context in which the Feingold diet was seen as a sensible alternative to stimulant drugs.¹⁰⁷³ The British BSE and Creutzfeldt-Jakob Disease (CJD) epidemic of the mid-1990s, which killed about eighty Britons and resulted in the culling of nearly five million cattle, for example, not only raised questions about how cattle were reared, but also about the safety of the food supply more generally. Not only did Britons increasingly consider organic food alternatives following the epidemic, but organic food activists also employed it to garner support for their cause.¹⁰⁷⁴

Similarly, concerns about genetically-modified foods, pesticides, food poisoning and diet-

¹⁰⁷³ For more on the origins of the British organic food movement, see: Philip Conford, 'The Alchemy of Waste: The Impact of Asian Farming on the British Organic Movement', *Rural History* 6 (1995), 103-14; Philip Conford, *Origins of the Organic Movement* (Edinburgh: Floris Books, 2001); Michael Brander, *Eve Balfour: Founder of the Soil Association and Voice of the Organic Movement: A Biography* (Haddington: Glenneil Press, 2003); David Matless, 'Bodies Made of Grass Made of Earth Made of Bodies: Organism, Diet, and National Health in Mid-Twentieth Century England', *Journal of Historical Geography* 27 (2003), 355-76; Richard Moore-Colyer, 'Towards "Mother Earth": Jorian Jenks, Organicism, the Right and the British Union of Fascists', *Journal of Contemporary History* 39 (2004), 353-71; Richard Moore-Colyer and Philip Conford, 'A "Secret Society"? The Internal and External Relations of the Kinship in Husbandry', *Rural History* 15 (2004), 189-206.

¹⁰⁷⁴ Kelly Morris, 'A Danger at My Table?', *Lancet* 354 (1999), 1565; Anonymous, 'Millions Turn to Organic Food', BBC News, 8 February 2000, www.news.bbc.co.uk/1/hi/health/634371.stm, accessed 13 March 2009; Anonymous, "'Growing Fears" over Mad Cow Disease', BBC News, 19 October 2000, www.news.bbc.co.uk/1/hi/scotland/979103.stm, accessed 13 March 2009; Karen Birchard, 'Europe Tackles Consumer Fears over Food Safety', *Lancet* 357 (2001), 1276; Rachel Schurman, 'Fighting "Frankenfoods": Industry Opportunity Structures and the Efficacy of the Anti-Biotech Movement in Western Europe', *Social Problems* 51 (2004), 243-68, at p. 254.

related chronic diseases, such as obesity and diabetes, combined with often enthusiastic reporting of such ‘food crises’, reduced British consumers’ faith in the food supply and made theories, such as Feingold’s, appear more plausible.¹⁰⁷⁵

Increased interest in the Feingold diet also paralleled other developments which highlighted British dissatisfaction with the state of the food supply. In 2005, for example, a documentary starring celebrity chef, Jamie Oliver, was broadcast on British television and documented the nutritional content of cafeteria food fed to children in a borough of London. Finding the food to be largely processed, lacking nutrition and high in saturated fat and chemicals, Oliver launched a campaign, called ‘Feed Me Better’, to provide additive-free, organic and seasonal food in school cafeterias and collected over 270 000 signatures on a petition that was delivered to Downing Street. Later that year, the Blair government pledged £280 million of support.¹⁰⁷⁶ On his website, Oliver listed ‘poor concentration’, ‘hyperactivity and behavioural problems’, and ‘mood swings’, as effects of the ‘processed junk foods’ served in schools, thus underlining a link between nutrition and mental health.¹⁰⁷⁷

Although the campaign was seen to be largely a success, it was also clear that there were many obstacles to changing how British children were fed, including financing such changes, educating parents, teachers, heads and school dinner ladies and, perhaps most importantly, convincing children to eat the healthier food.¹⁰⁷⁸ Oliver even backed a plan proposed by a school in North Wales to lock school gates at lunch time in order to prevent children from

¹⁰⁷⁵ For an insider’s perspective on how the media cover food scares, see: Nicola Carlsaw, ‘Communicating Risks Linked to Food – The Media’s Role’ *Trends in Food Science and Technology* 19 (2008), S14-S17. Recent popular explorations of the issue of food safety and processed food include Eric Schlosser, *Fast Food Nation: The Dark Side of the All-American Meal* (New York: Houghton Mifflin, 2001); Hugh T. Pennington, *When Food Kills: BSE, E. coli, and Disaster Science* (Oxford: Oxford University Press, 2003); Morgan Spurlock, *Supersize Me* (Kathbur Pictures, 2004).

¹⁰⁷⁶ Channel 4, *Jamie’s School Dinners*, www.channel4.com/life/microsites/J/jamies_school_dinners/index.html, accessed 13 March 2009.

¹⁰⁷⁷ Jamie Oliver, www.feedmebetter.com/why/junkfood.html, accessed 10 October 2007.

¹⁰⁷⁸ Anonymous, ‘Oliver’s School Meal Crusade Goes On’, BBC News, 4 September 2006, www.news.bbc.co.uk/1/hi/uk/5313882.stm, accessed 13 March 2009.

leaving school grounds to buy junk food.¹⁰⁷⁹ Nevertheless, the ‘Feed Me Better’ campaign indicated how not only the British public, but also their government, was willing to make nutrition a public health policy priority.

In addition, many large companies in the British food industry decided to pre-empt legislative action and changed the production and packaging of their products to accord with such nutrition concerns. Although some of the measures were designed to avoid accidental allergic reactions to peanuts and other food allergens, many addressed fears about food additives, particularly colours. Marks and Spencer, for example, removed 99 per cent of the artificial colours and flavours found in their foods, and other supermarkets followed suit.¹⁰⁸⁰ Nestlé also pledged to remove all artificial colours from Smarties, resulting in the demise of the blue Smartie, and Burton Foods, the makers of Jammie Dodgers, followed suit.¹⁰⁸¹ Other food manufacturers and supermarkets, often responding to pressure from parents, also began to use natural dyes, such as beetroot, instead of those made from petrochemicals, and emphasised on the packaging of certain products that they were free of artificial additives.¹⁰⁸² Such actions, which did not occur to the same extent in the United States, indicate how a combination of celebrity-driven publicity, parent pressure and the findings of medical research created an environment in which it was feasible to change the production, packaging and marketing of food products in an effort to reduce the amount of artificial additives.¹⁰⁸³

¹⁰⁷⁹ Nigel Bunyan, ‘Jamie Oliver Supports School Lunch Lock-Ins’, *Daily Telegraph*, 6 September 2007, www.telegraph.co.uk/news/main.jhtml;jsessionid=AOOQCMF2RSGZJQFIQMGCF4AVCBQUIV0?xml=/news/2007/09/06/njamie106.xml, accessed 13 March 2009.

¹⁰⁸⁰ Anonymous, ‘Food Watchdog Condemned’.

¹⁰⁸¹ Elliot, ‘Food Alert’.

¹⁰⁸² Anonymous, ‘Food Watchdog Condemned’.

¹⁰⁸³ Although American supermarkets have not been as responsive as their British counterparts in switching to natural dyes, a number of organic supermarkets, such as Trader Joes and Whole Foods Market, have become more predominant in the United States, providing American consumers with additive-free products. Many of the Feingold families interviewed reported relying almost exclusively on these supermarkets for their groceries, even though a visit to one of these supermarkets could require an extra hour of travel time and cost up to a third extra. Paula Kimball, Telephone Interview, 4 February 2008; Taunya Stephenson, Telephone Interview, 1 April 2008; Lisa Manciewicz, Telephone Interview, 17 April 2008; Brian Rigg, Telephone Interview, 19 May 2008.

Running parallel to fears about the food supply were concerns about pharmaceutical products which, in turn, led parents in the United States, Britain and elsewhere to consider alternative treatments for hyperactivity. As Chapter 6 contends, American parents were worried about the use of stimulant drugs as early as the late 1960s and this was one of the chief reasons why they turned to alternatives such as the Feingold diet. Nevertheless, prescriptions for drugs such as Ritalin continued to increase in the decades that followed. During the 1990s and 2000s, however, a number of reports emerged which raised suspicions about the makers of hyperactivity drugs, as well as the pharmaceutical industry in general. In 1996, for example, CNN.com reported that scientists had shown how Ritalin caused cancer in mice.¹⁰⁸⁴ A year later the CBC described how scientists at a NIH conference which addressed the use of Ritalin ‘expressed concerns about a lack of data on the long-term effects of the drug’.¹⁰⁸⁵

The short-term health risks of Ritalin also made headlines during the late 1990s when the American Heart Foundation issued guidelines for the monitoring of children prescribed Ritalin and other stimulants after a number of children and adolescents taking such drugs died suddenly from cardiovascular problems.¹⁰⁸⁶ One well-publicised case, for example, was that of Matthew Smith, a fourteen-year-old whose fatal heart failure was attributed to long-term Ritalin use.¹⁰⁸⁷ Although the percentage of children thought to be at risk from sudden death caused by stimulants was thought to be low, health authorities in North America were

¹⁰⁸⁴ Dan Rutz, ‘Ritalin Comes Under Scrutiny After Cancer Found in Mice’, www.edition.cnn.com/HEALTH/9601/ritalin/index.html, 14 January 1996, accessed 20 March 2009.

¹⁰⁸⁵ Anonymous, ‘Hyperactivity Drug Under Scrutiny’, 18 November 1998, www.cbc.ca/health/story/1998/11/18/ritalin981118a.html

¹⁰⁸⁶ Steve Salvatore, ‘Group Issues Guidelines for Monitoring Ritalin in Children’, 9 November 1998, www.edition.cnn.com/HEALTH/9811/09/hearts.ritalin/index.html, accessed 20 March 2009.

¹⁰⁸⁷ Fred A. Baughman, Jr., *The ADHD Fraud: How Psychiatry Makes ‘Patients’ out of Normal Children* (Victoria, BC: Trafford Publishing, 2006), 1. Other popular books criticising the notion of hyperactivity proliferated during the 1990s and 2000s. For example: Peter Breggin, *Talking Back to Ritalin: What Doctors Aren’t Telling You About Stimulants for Children* (Monroe, ME: Common Courage Press, 1998); Lawrence H. Diller, *Running on Ritalin: A Physician Reflects on Children Society, and Performance in a Pill* (New York: Bantam Books, [1998] 1999); Richard J. DeGrandpre, *Ritalin Nation: Rapid-Fire Culture and the Transformation of Human Consciousness* (New York: W. W. Norton: 1999).

alarmed nonetheless. Health Canada, for example, temporarily removed the hyperactivity drug Adderall from the marketplace in 2005 after reports of twenty deaths.¹⁰⁸⁸ Although the American FDA did not follow suit, an FDA advisory committee recommended in 2006 that guidelines and warnings be strengthened to reflect such risks. Steven E. Nissen, a consultant for the advisory committee, believed that the risk of serious cardiovascular problems ‘warranted strong and immediate action’ and argued that hyperactivity drugs be subject to ‘more selective and restricted use’.¹⁰⁸⁹ In response, the FDA ‘directed the manufacturers of all ADHD medicines to add a “black box” warning to their products, pointing to the potential cardiovascular risks’.¹⁰⁹⁰

Concerns about hyperactivity drugs were matched by concerns about the side effects of other drugs, as well as the motives of the pharmaceutical industry in general. While the cardiovascular health risks of pain reliever Vioxx led to American Senate investigations, anti-depressant Zoloft was linked to suicidal and homicidal behaviour.¹⁰⁹¹ In Britain a report from the University of Hull announced in 2008 that the commonest anti-depressant drugs, including Prozac, had little effect in all but the most severely depressed patients, partially prompting Health Minister Allan Johnson to announce plans to train 3,600 more therapists able to provide ‘talk therapy’, rather than drugs.¹⁰⁹²

¹⁰⁸⁸ Thomas E. Wilens, Jefferson B. Prince, Thomas J. Spencer and Joseph Biederman, ‘Stimulants and Sudden Death: What Is a Physician to Do?’ *Pediatrics* 118 (2006), 1215-19, at p. 1215; Iverson, *Speed, Ecstasy, Ritalin*, 64.

¹⁰⁸⁹ Steven E. Nissen, ‘ADHD Drugs and Cardiovascular Risk’, *New England Journal of Medicine* 354 (2006), 1145-8, at p. 1447-8.

¹⁰⁹⁰ Iverson, *Speed, Ecstasy, Ritalin*, 64. The risks were believed to be higher if children were engaged in strenuous exercise; indeed, Matthew Smith was skateboarding when he suffered heart problems. Ironically, exercise is thought to be one of the ways in which children can burn off excess energy that might exacerbate their hyperactivity. Duffy, ‘Hyperactive Children “Need Exercise, Not Drugs”’, 27.

¹⁰⁹¹ Elaine Cassel, ‘Did Zoloft Make Him Do It?’, 7 February 2005, www.edition.cnn.com/2005/LAW/02/07/cassel.pittman/index.html, accessed 20 March 2009; Lou Dobbs, ‘Promote Safety not Profits’, 14 January 2005, www.edition.cnn.com/2005/US/01/13/fda.safety/index.html, accessed 20 March 2009.

¹⁰⁹² Anonymous, ‘Anti-Depressants, Little Effect’, 26 February 2008, www.news.bbc.co.uk/1/hi/health/7263494.stm, accessed 20 March 2009; Irving Kirsch, Brett J. Deacon, Tania B. Huedo-Medina, Alan Scoboria, Thomas J. Moore and Blair T. Johnson, ‘Initial Severity and Anit-Depressant Benefits: A Meta-Analysis of Data Submitted to the Food and Drug Administration’, *PLoS Medicine* 5 (2008), 260-7, at p. 260.

Perhaps the most extreme example of public distrust of drug companies and conventional medical knowledge, however, was the furore over the measles, mumps and rubella vaccine (MMR) that emerged during the late 1990s when British surgeon Andrew Wakefield (b. 1957) alleged a possible connection between MMR and autism.¹⁰⁹³ Although Wakefield continued to advise parents to vaccinate their children against measles, mumps and rubella using single, separately-administered vaccines, he nonetheless suggested that MMR itself could be a cause of autism and, as did Feingold, voiced his concerns at a press conference before he had gathered much supporting evidence.¹⁰⁹⁴ Many other features of the debates about MMR help to explain why Feingold's theory also experienced a renaissance during the same period.¹⁰⁹⁵

As with the debates about Feingold's idea, the controversy that erupted over MMR pitted the majority of the medical profession against parents and anti-vaccination activists, as well as a small number of unorthodox medical professionals. Whereas the risks of employing the Feingold diet were comparably non-existent, however, concern about MMR resulted in not only a decline in the use of the vaccine, but also an increase in cases of measles in both Britain and North America.¹⁰⁹⁶ Despite the emergence of little scientific evidence to support

¹⁰⁹³ A. J. Wakefield, S. H. Murch, A. Anthony, J. Linnell, D. M. Casson, M. Malik, M. Berelowitz, A. P. Dhillon, M. A. Thomson, P. Harvey, A. Valentine, S. E. Davies and J. A. Walker-Smith, 'Ileal-Lymphoid-Nodular Hyperplasia, Non-Specific Colitis, and Pervasive Developmental Disorder in Children', *Lancet* 351 (1998), 637-41.

¹⁰⁹⁴ Harry Collins and Trevor Pinch, *Dr. Golem: How to Think About Medicine* (Chicago: University of Chicago Press, 2005), 186.

¹⁰⁹⁵ Indeed, a number of the parents interviewed about the Feingold diet expressed concern about the vaccine, including: Bonnie Kowaliuk, Telephone Interview, 5 November 2007; Anonymous, 26 January 2008; Shula Edelkind, Telephone Interview, 28 January 2008; Leah Hause, Telephone Interview, 12 February 2008; Lynn Murphy, Email Interview, 24 July 2008.

¹⁰⁹⁶ Anonymous, 'Measles Fears Prompt MMR Campaign', 7 August 2008, www.news.bbc.co.uk/1/hi/health/7545151.stm, accessed 23 March 2009; Jessica Snyder Sachs, 'Vaccines: Separating Facts from Fiction', 9 December 2008, www.edition.cnn.com/2008/HEALTH/family/11/05/par.vaccine.kids/index.html, accessed 23 March 2009; Madison Park, 'Autism Ruling Fails to Convince Many Vaccine-Link Believers', 14 February 2009, www.edition.cnn.com/2009/HEALTH/02/12/court.autism.reactions/index.html, accessed 23 March 2009; Stephen Strauss, 'Linking Vaccines, Autism Tantamount to Crying "Fire" When There Isn't One', 12 March 2009, www.cbc.ca/technology/story/2009/03/11/f-strauss-autism-vaccines.html, accessed 1 April 2009.

Wakefield's association of MMR and autism, and the fact that the public health risks of decreased vaccination could be severe, many parents grew alarmed that in attempting to prevent one disease they might trigger another, perhaps even more frightening, affliction. Nevertheless, even if there was an association between MMR and autism, it could be argued that the dangers posed by a widespread measles epidemic, for example, outweighed the risks of increased cases of autism.¹⁰⁹⁷ Perhaps the decision made by many parents to accept the greater likelihood of their child contracting measles, a potentially fatal disease, in the belief that this would prevent their child from developing autism, also indicated how fear of infectious disease in developed nations had been replaced by fear of chronic diseases and, especially, mental illness.¹⁰⁹⁸

Public fears about MMR reflected doubts about the safety of pharmaceutical products, but they also showed how concern could be stoked by the media and activist groups through the internet, the medium through which many Feingold families during the late 1990s and onward found out about the Feingold diet. For Feingold families the internet provided not only practical advice and information, including increasingly long lists of Feingold-friendly products, but it also afforded moral support for parents in the form of online support forums and chat rooms. Similarly, by 2002 there were twenty-two websites promoting the anti-MMR campaign, and the internet also became the medium through which parents could research MMR, analyse the countless debates, perhaps contributing to them themselves, and

¹⁰⁹⁷ Collins and Pinch describe this issue as the 'prisoner's dilemma': 'Think of vaccination as equivalent to a year in prison and catching the disease in question as equivalent to ten years. If everyone vaccinates, then everyone gets one year. If no one vaccinates, then everyone gets ten years. If everyone else vaccinates and you do not, you go free.' Ironically, the resolution of this ethical quandary threatened the previously successful writing partnership of Collins and Pinch, when Pinch decided to hold off vaccinating his child against whooping cough (pertussis) using the conventional American vaccine in order to obtain what he believed to be a safer vaccine from Japan. Collins disagreed with Pinch's rationale for this decision and their resulting debate is described at length in *Dr. Golem*. Collins and Pinch, *Dr. Golem*, 192-201.

¹⁰⁹⁸ The notion that the spectre of infectious disease had been replaced by the threat of mental illness was expressed as early as 1963 by President Kennedy in a speech to Congress on mental illness and retardation. Certainly measles remains a major public health risk in the developing world and in countries, such as Japan, where vaccination uptake is relatively low. Kennedy, 'Message from the President', 729; Michael Fitzpatrick, *MMR and Autism: What Parents Need to Know* (New York: Routledge, 2004), 1.

make decisions about their children's vaccination.¹⁰⁹⁹ In Britain, where the MMR controversy was most vociferous, primarily due to the fact that vaccination was not compulsory as it was in the United States, the public's lack of confidence in what the medical profession, the pharmaceutical industry and the government had to say about MMR also reflected the perception that these authorities had recently made mistakes with respect to other public health controversies, most notably the BSE outbreak.¹¹⁰⁰

That parents in both North America and Britain were convinced to forego MMR vaccination, despite the fact that doing so was unsupported by scientific evidence, was possibly unethical and left their children vulnerable to previously widespread and potentially fatal infectious diseases, highlighted the apprehension with which many people viewed medicine, the industries associated with it and the governing bodies designed to regulate it during the late 1990s and 2000s. In the midst of this anxiety, it is not surprising that parents and researchers, particularly those in Britain, also considered alternatives to conventional hyperactivity treatment. Indeed, although the Feingold diet became the most discussed alternative, parents at the turn of the millennium also considered others, including fish oils, biofeedback and even increasing the amount of play-time allowed to their children.¹¹⁰¹ Just as conditions were appropriate for parents and physicians to consider the Feingold diet in the United States during the 1970s, they were ripe for a resurgence of interest in the diet during the 2000s. Whether this shift in opinion will result in a watershed in how hyperactivity is researched,

¹⁰⁹⁹ Collins and Pinch, *Dr. Golem*, 185.

¹¹⁰⁰ Collins and Pinch, *Dr. Golem*, 189; Tammy Boyce, *Health, Risk and News: The MMR Vaccine and the Media* (New York: Peter Lang Publishers, 2007), 1. The vitriolic nature of the British debate about MMR is evident in the first pages of Richard Horton's book on the subject. Horton, currently the editor in chief of the *Lancet*, participated in the debates himself, and described what followed Wakefield's press conference as 'a cascade of bizarre and catastrophic events'. Richard Horton, *MMR: Science and Fiction: Exploring a Vaccine Crisis* (London: Granta Books, 2004), 1-7.

¹¹⁰¹ Anonymous, 'The Importance of Not Being Earnest', 6 November 1998, www.news.bbc.co.uk/1/hi/health/208729.stm, accessed 23 March 2009; Rita Baron-Faust, 'Biofeedback Widens its Role in Medicine', 17 February, 2000, www.edition.cnn.com/2000/HEALTH/alternative/02/17/neuro.feedback.wmd/index.html, accessed 23 March 2009; Anonymous, 'Diet Can Ease Behaviour Problems', 13 February 2002, www.news.bbc.co.uk/1/hi/health/1816938.stm, accessed 23 March 2009.

understood and treated, however, remains to be seen, and will similarly depend on a wide range of cultural, economic, political and scientific factors. Although a recent German report associating hyperactivity and eczema suggests that the link between allergic disease and behavioural disorders is becoming more concrete, increased unemployment might convince a wider range of people to try amphetamines in an effort to improve their vocational performance, and lead to renewed acceptability for their use in children.¹¹⁰² The reasons for the ultimate acceptance or rejection of the Feingold diet, or, possibly more likely, its continued positioning on the margins of medicine, will be determined not only by double-blind trials, but also by how well it accords with the prevailing scientific trends, societal conditions and ideological beliefs.

This thesis has contended that, in order to comprehend the changing fortunes of Feingold's hypothesis and, by extension, the fate of other controversial medical ideas, it is essential to analyse the broader historical contexts into which such ideas emerged. The corollary to this argument is that current medical debates, including the ongoing arguments about the Feingold diet, can and, perhaps, should be informed by discussions about the histories of these debates. It is important to understand, however, that historical context is not a fixed, indivisible concept that can be easily ascertained. Part of the reason why the Feingold diet was so contested was that, while it appeared logical to some sectors of the American population, especially parents who were frustrated with conventional hyperactivity treatments, it seemed suspect to others, most notably psychiatrists who had recently resolved their own debates about the aetiology of hyperactivity and orthodox allergists who had long held suspicions about food allergy. Feingold's partially successful attempt to present the

¹¹⁰² Linda Ciampa, 'Ritalin Abuse Scoring High on College Illegal Drug Circuit', 8 January 2001, www.edition.cnn.com/2001/HEALTH/children/01/08/college.ritalin/index.html, accessed 23 March 2009; Leslie Iverson, *Speed, Ecstasy, Ritalin: The Science of Amphetamines* (Oxford: Oxford University Press, [2006] 2008), 71-3, 104-5; Anne Harding, 'Study: Kids with Eczema More Likely to Have ADHD', 18 February 2009, www.edition.cnn.com/2009/HEALTH/02/18/healthmag.eczema.adhd/index.html, accessed 23 March 2009.

origins of his diet in *Why Your Child is Hyperactive* in a manner that would be palatable to both conservative physicians and desperate parents suggests that, as a previously orthodox paediatric allergist, he understood that his theory had to operate under multiple paradigms at once.

Similarly, the way in which word about the Feingold diet spread from San Francisco to the rest of the United States and elsewhere also spotlights the gaps between popular and medical understandings and concerns about food additives and hyperactivity. Although Feingold's intent was to publish his theory conventionally, in medical journals, such as *JAMA*, the medical establishment, wary of associating themselves with such a volatile claim, rebuffed his overtures. In contrast, the American media and the general public found Feingold's hypothesis to address both dissatisfaction with conventional explanations and treatments of hyperactivity and worries about food additives, providing him with a forum from which to present his ideas to a wider and more sympathetic audience. This shift in focus, from trying to convince orthodox physicians to concentrating on parents, the media and the general public, had crucial ramifications for the fate of the Feingold diet; while it alienated many physicians, it empowered Feingold families and FAUS to become medical experts in their own right, and ensured the survival of Feingold's theory long after Feingold's death and the subsequent dwindling of medical research into his idea.

Finally, in order to understand why different parties, including the media, physicians and parents, made decisions about the efficacy of the Feingold diet, it is vital to analyse carefully the basis on which such opinions were formed. While the judgements of both Feingold's supporters and detractors in the media and the medical community were characterised by prejudice - with some notable exceptions, including Jean Mayer, C. Keith Connors and Bonnie Kaplan - Feingold families were more concerned with analysing whether or not the

diet worked for them. And it must be said that the diet did work for most of them. Given this, and the positive results from the Southampton studies, the role of parents, as well as their children, in the resolution of medical debates is a subject that requires further study from medical historians, medical researchers and health policy makers.

Although it has been commonly asked, the question at the heart of this thesis has not been: ‘Did the Feingold diet work?’ Although this is an important inquiry, and one that can be confidently answered in the affirmative with respect to many of the Feingold families, it is one that requires deconstruction. What is meant by ‘the Feingold diet’? Is it simply a list of acceptable foods or, instead, a broader lifestyle choice that encompasses not only dietary change, but also a family’s determination to understand their children’s behaviour in a broader social, educational, emotional and ecological context, and respond to it accordingly? If so, does it not also imply that parents take up the role of medical experts, not only becoming canny observers and experimenters, but also the arbiters of their children’s health care? Moreover, what exactly is meant by ‘work’? Did it work for every family, at every time, in every circumstance? The answer to this is clearly no, but when the reasons why the diet was not effective are analysed, it becomes evident that many other factors, including availability of Feingold-friendly products, support from school authorities, medical professionals, family and friends as well as the willingness of both children and parents to persevere on the diet, must be also considered. Addressing whether the Feingold diet worked or not is a complicated task and, although opinions about food additives and hyperactivity are changing somewhat, there are still numerous barriers in place that will hamper many families’ attempts to employ it successfully.

Instead of asking ‘did the Feingold diet work?’, this thesis has questioned why the efficacy of the Feingold diet became such a divisive subject and what factors led to such debate. What is

revealed in the answers to this question is that medical controversies are about more than academic debates over matters of scientific truth. The points of view represented on various sides of the debate also reflect the ideals, desires, experiences and beliefs of those who hold them. To a certain degree, there is nothing wrong with this and, regardless, it is probably impossible to filter such factors out of any medical controversy. But what is problematic is that when such debates have occurred, the opinions and experiences of patients and their families, as well as unorthodox medical professionals, have been downgraded and ignored, attenuating any resolution that has eventually emerged. This does not imply that unorthodox or popular beliefs should not be rigorously critiqued. In the case of MMR, it is highly probable that the medical authorities were correct that the link between the vaccine and autism was tenuous and that, even if a link could be proven, the morality of refusing vaccination was questionable. But during debates about the Feingold diet, the experiences of parents were overlooked as physicians tallied, not particularly carefully, the results of double-blind controlled trials. As the Feingold diet once again becomes a contentious issue, perhaps physicians and policy makers will take note of the history of the initial debates and reconsider how they judge the opinions and experiences of patients and their families.

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